1959

Eugenics Quarterly

Vol. 6 June, 1959 No. 2

PROCEEDINGS

International Conference

on

DIFFERENTIATION IN CURRENT MATING AND FERTILITY TRENDS

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ON

DIFFERENTIATION IN CURRENT MATING AND FERTILITY TRENDS

Held on

Saturday, February 14, 1959

at

THE NEW YORK ACADEMY OF MEDICINE BUILDING

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For U.S.A.: Albert J. Phiebig, P.O. Box 352, White Plains, N.Y.

Eugenics Quarterly

Published by the AMERICAN EUGENICS SOCIETY, Inc.
230 Park Avenue, New York 17, N. Y.

Vol. 6, No. 2 June, 1959

DIFFERENTIATION IN CURRENT MATING AND FERTILITY TRENDS

Program Chairman: FRANZ J. KALLMANN, M.D., New York State Psychiatric Institute

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Vol. XII, No. 3 March 1959

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THE EUGENICS REVIEW

Volume 51, Number 1 April, 1959

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Morning Session

GENERAL MATING AND FERTILITY TRENDS

Chairman: FREDERICK OSBORN, The Population Council

CURRENT MATING AND FERTILITY PATTERNS AND THEIR DEMOGRAPHIC SIGNIFICANCE

CLYDE V. KISER
Milbank Memorial Fund

PURPOSE AND SCOPE OF PAPER

The purpose of this paper is to describe recent trends in, and current patterns of, mating and fertility in modern Western countries. Those responsible for planning this Conference apparently felt that a paper on this topic might provide something of a norm or benchmark by which to judge the findings of the subsequent papers on the mating and fertility behavior of persons with various types of psychiatric problems.

Inasmuch as the existing data on fertility of these special groups are restricted largely to the more modernized countries, the present paper will also be so restricted. Thus the paper is not concerned with the high and uncontrolled fertility of the underdeveloped countries. More specifically it will be concerned mainly with the experience of the United States, Canada, several countries of Europe, and Australia. The source materials are mainly from the United Nations and the United States government agencies (8-15 incl.).

MATING

We shall, for present purposes, regard mating as synonymous with marriage. In Western societies probably most of the mating is within marriage. It is acknowledged that according to studies by Kinsey and others premarital and extra-marital mating are not uncommon. According to the National Office of Vital Statistics about one-fifth of the live births among nonwhites in the United States are illegitimate (12, Table AR). In certain Scandinavian countries apparently a rather considerable proportion of first births are premaritally conceived even though they are legitimized by marriage.

It is also acknowledged that there may be some relationship, both selective and

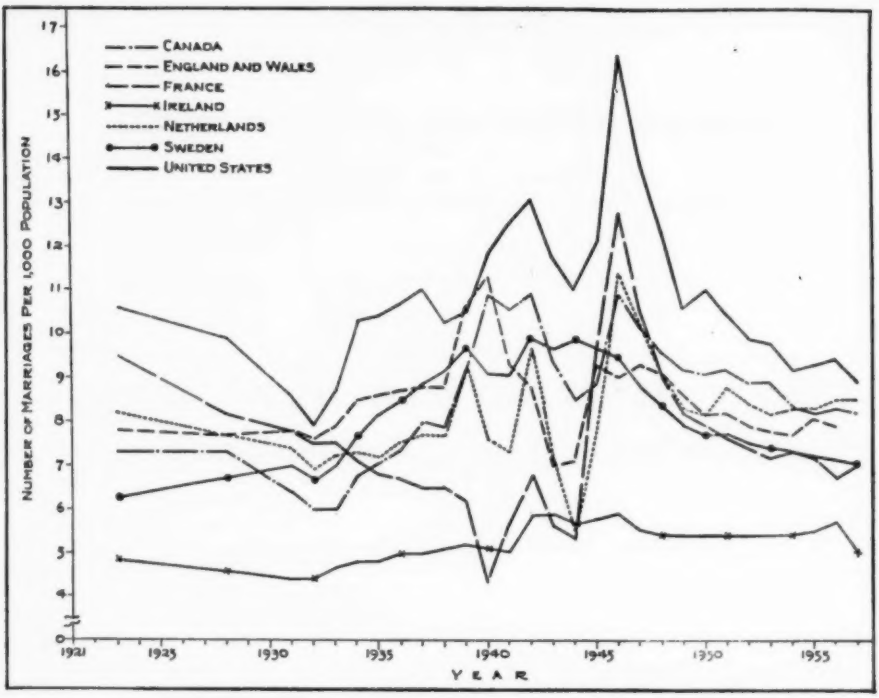

FIGURE 1. Annual number of marriages per 1,000 population in selected countries, 1921-1957. (Adapted from various issues of *Demographic Yearbook* and *Population Index*, References 5 and 13).

determinative, between certain types of mental illness and extra-marital mating. However, the writer knows of no adequate data on this question.

TRENDS OF MARRIAGE RATES

Figure 1 depicts trends in crude marriage rates in selected industrialized countries since 1921. It will be noted that throughout the whole period considered the rates for the United States are relatively high and those for Ireland are conspicuously low. Among the factors contributing to the relatively high marriage rate in the United States may be a somewhat younger age distribution, the relatively high proportions rural, and the relatively high proportions that are nonwhite. Also, to a minor extent the relatively high rate of divorce itself augments the marriage rate of the United States.

As for Ireland, abstention from marriage and late age at marriage apparently are forms of population control that the Irish have imposed upon themselves. Unforgettably subjected to the potato famine and the ensuing misery of the 1850's, the Irish nevertheless apparently have remained firm in their religious opposition to contraception. At first, migration to the United States provided some relief from population pressure. When this outlet became closed, the

Malthusian factor of moral restraint apparently became the accepted means of population control. Some of the unmarried youngsters migrate to England and other countries for temporary or permanent residence. Of those that remain at home a few marry at young ages. Probably many mature men and women who are more or less formally engaged live with their aged parents and wait around until the circumstances are such that they can be married. Probably for many the opportunities and even the inclinations fade with age.

As for trends, most of the Western countries experienced declines in the marriage rate during the late twenties and early thirties. In the United States the crude marriage rate dropped to 7.9 per 1,000 population in 1932, the lowest point on record. Apparently during this period thousands of marriages were cancelled or postponed because of the economic depression. No job, no money, no marriage.

Following its low point of 1932, the marriage rate climbed slowly with the gradual economic recovery. The threat of war in 1938, and the European outbreak of war in 1939 stimulated defense spending and the creation of new jobs in the United States. Defense production was stepped up further during the "arsenal of democracy" era, and it became an all-out effort when we entered the war in December, 1941.

Besides the economic factors, the entrance of the United States into the war stimulated marriages and births directly. Young men sometimes sought marriage and parenthood to avoid or to defer conscription into military service. The monthly marriage rates indicate a rush to the altar right after Pearl Harbor. Ten months after Pearl Harbor there was a sharp peak in the monthly birth rate.

On an annual basis, the marriage rate in the United States reached a wartime peak of 13.1 in 1942. It dropped to 11.0 in 1944 when a large number of soldiers were overseas. With the ending of the war in 1945 and the ensuing demobilization, the year 1946 marked the highest marriage rate on record in this country, 16.4 marriages per 1,000 population. The European countries exhibited peaks in marriage rates in 1946, but none equaled the magnitude of that in the United States. Even Ireland, neutral in the war, exhibited a slight rise in 1946. In most of the countries considered the trend of the marriage rate has been downward since 1946. In 1957, the marriage rates varied from 5 in Ireland to 8.9 in the United States.

AGE AT MARRIAGE

There has been a marked trend toward *younger* marriages in recent years. Computations based upon annual registration data may sometimes conceal or minimize this trend because of counteracting increases in remarriages and because of the aging of the population. For this reason, students frequently use census data on marital status by age for computing average or median ages at marriage, as in Figure 2.

For the countries depicted in Figure 2, the median age at marriage around 1950 was lowest in the United States and highest in Ireland. The median age at marriage for women in the United States was 21.3 years in 1930 and 20.2 years in 1950, a decline of 1.1 years. In Ireland the median was 27.7 in 1941 and 26.0 in 1951, a decline of 1.7 years.

The trends and differentials in marriage patterns are depicted much more strikingly in Figure 3 which is concerned with percentages ever married among

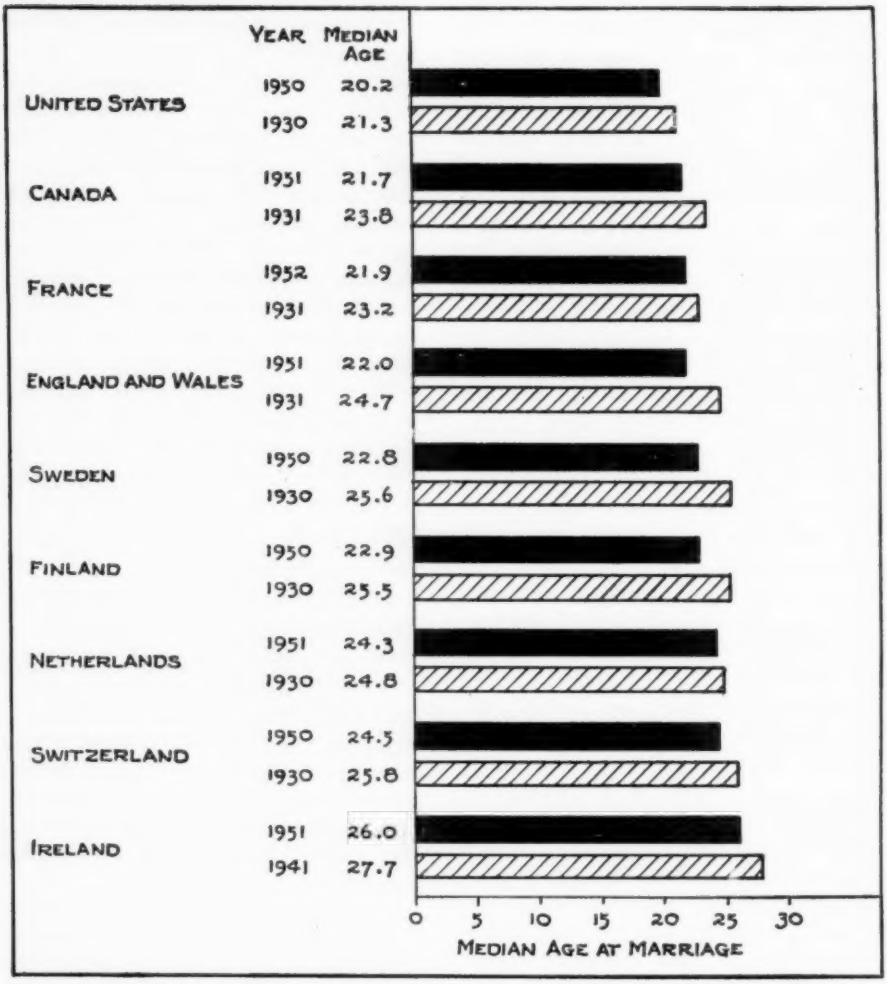

FIGURE 2. Median age at first marriage in selected countries for hypothetical birth cohorts of women marrying under age 45 based on proportions of women ever married at various ages around 1930 and 1950. (Reference 15, p. 54.)

women 20-24 years old in the various countries for the approximate years 1940 and 1950. Thus in 1940 only slightly over half of the women 20-24 years old in the United States had ever married. In 1950, the proportion was slightly over two-thirds. Other data indicate that the percentage was 71 for 1957 (2, p. 45). In Ireland only about 12 per cent of the women 20-24 were married in 1941, and the corresponding percentage for 1951 was about 18.

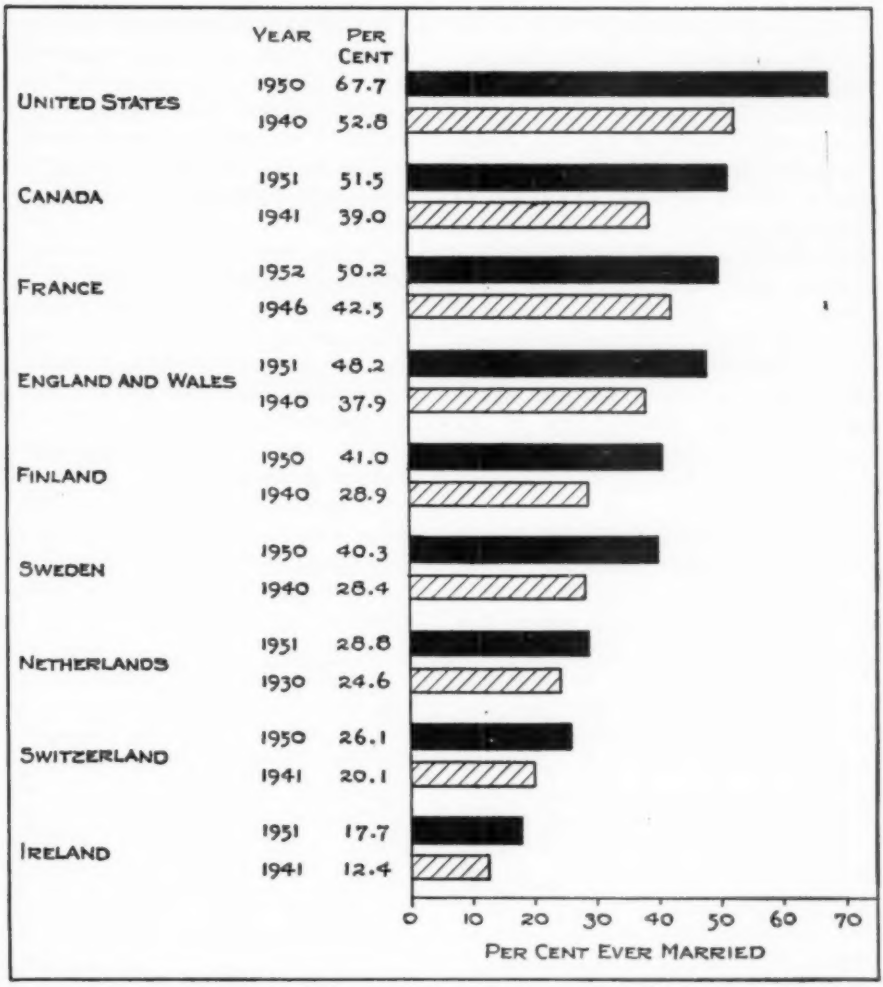

FIGURE 3. Percentages ever married among women 20-24 years, for selected countries and years *circa* 1940 and 1950. (Reference 15, p. 49.)

CONTRACEPTIVE PRACTICE

The practice of contraception is widely prevalent in most of the modernized countries. It has implemented the long-standing decline in size of completed family. It is the means by which couples plan or attempt to plan the number and spacing of their children, and as such it is an important aspect of the patterns of mating and fertility in modern countries. Among the couples in the Indianapolis Study about 89 per cent reported at least some experience with contraception when they were interviewed in 1941.

Virtually all, about 98 per cent, of the couples classified as "relatively fecund" reported some previous use of contraception (16).

A study "Growth of American Families" conducted jointly by the Survey Research Center of the University of Michigan and the Scripps Foundation for Research in Population Problems was carried out in 1955 among some 2,700 white couples with wife 18-39 years of age. This study collected data on number of children born to date, total number of children desired, and past contraceptive practice. The findings of this study are now being published in a book by Whelpton, Freedman, and Campbell (1).

In a recent report of some of the materials Dr. Ronald Freedman stated, "The great majority of American couples in the childbearing years report that they have used contraception already or that they intend to do so in the future. The exceptions are mainly couples who have no need or little need for contraception because they are subfecund. Among fecund couples in our sample, 83 per cent reported that they had already used contraception and an additional 7 per cent intended to do so after one or more pregnancies" (2). Some of the nonusers were recently married couples who wanted one or two children soon after marriage, and others just had not gotten around to starting preventive measures.

About 19 per cent of the couples in the Growth of American Families study had *Completely Planned* fertility in that they had no pregnancies except those that were deliberately planned by stopping contraception in order to conceive. About 66 per cent had *Partially Planned* fertility. ("Some of their pregnancies may have been planned, some may have been accidents, or contraception may never have been used, but none of these couples have had unwanted children.") About 13 per cent were *Excess-Fertility* couples in that the last pregnancy was not wanted by either the wife or husband.

A companion study of the "Growth of American Families" is the Study of the Future Fertility of Two-Child Families being carried out under the technical direction of the Office of Population Research, Princeton University and under the sponsorship of the Milbank Memorial Fund. It is a partially longitudinal study of 1,165 native-white couples in seven metropolitan areas of the United States who had their second child in September, 1956. These couples were visited for the first time in the spring of 1957. They will be revisited in the spring of 1960.

According to data secured from the first visits, about 59 per cent of the couples were classified as "successful planners" in that they had planned the second child. The proportion of successful planners varied by religion of the couple. It was lowest (48-50 per cent) for the Catholics and for the mixed marriages solemnized by a priest. It was highest (83 per cent) for the Jewish couples, and intermediate (58 per cent) for Protestant couples (6).

CRUDE BIRTH RATE

The trends of crude birth rates for selected modern countries are presented in Figure 4. For most of the countries and years considered, Canada had the highest birth rate. Since 1949 the order has been, Canada, United States, Netherlands, Ireland, France, and (interchangeably in lowest position) England and Wales, and Sweden.

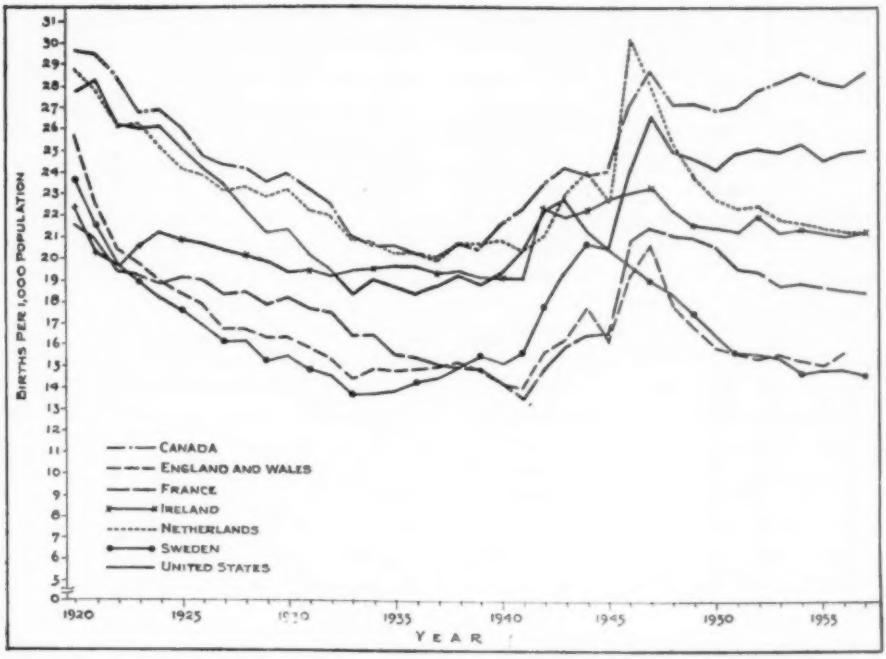

FIGURE 4. Crude Birth Rates. Annual number of live births per 1,000 population in selected countries, 1920-1957. (Reference 15, p. 130, and reference 12, Table AE.)

As with marriage rates, the crude birth rates for most of the countries considered reached low points during the middle thirties. The exceptions were France which exhibited a decline through 1941 and Ireland which exhibited rather uniform birth rates througout the thirties. Five of the countries exhibited peak birth rates in 1947. The peak came in 1946 for Netherlands and in 1944 for Sweden. The nature of the experience of these countries in the Second World War helps to explain the timing of the peak birth rates.

There has been a strong persistence of the high birth rate in Canada and the United States. In 1957 the rates were 28.6 for Canada and 25 for the United States. However, in Sweden and England and Wales, the crude birth rates are now almost as low as they were during the depression.

The trends in age-specific *general* fertility rates are presented in Figure 5 for selected countries. These are annual numbers of births per 1,000 women of specific age regardless of marital status. It will be noted that in recent years the United States has stood at the top and Canada in second place with reference to fertility rates among women under 25.

The relatively high rates for United States women under 25 in recent years doubtless is due in large part to the relatively high proportions ever married.

At older ages, the fertility rates for

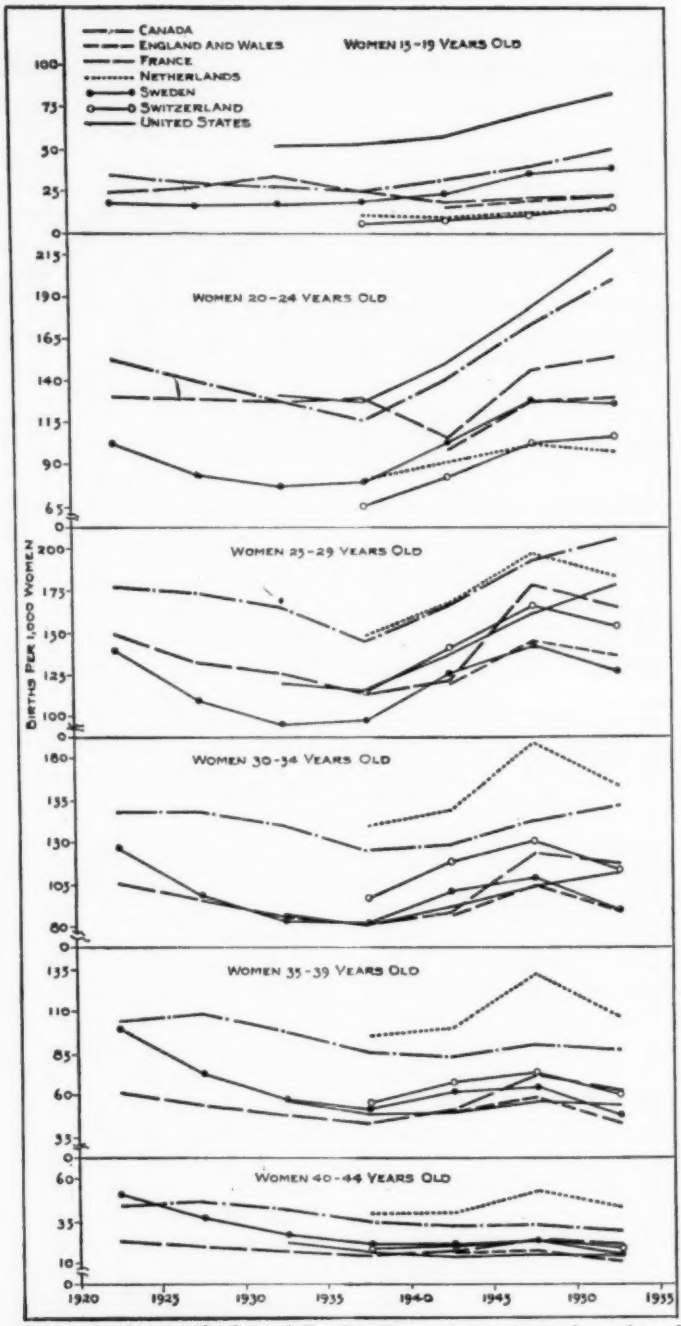

FIGURE 5. Age-specific General Fertility Rates. Average annual number of live births per 1,000 women of specified age in selected countries for five-year periods during 1920-1954. (Reference 15, pp. 134-135.)

United States women are relatively low in comparison with those of other countries. Except for Netherlands the postwar baby boom apparently brought little increase to fertility rates of women 35 years of age and over.

Figure 6 brings together the data for women of successive ages of the childbearing span for the various countries and for the period 1950-1954. This points up again that the fertility of the United States women in comparison with that in other countries stands highest at ages under 25 but relatively low at ages 30-44. The fertility of Netherlands women stands lowest at ages under 25 and highest at ages 30-44. The fertility rate for Canadian women surpasses that of the other countries considered at ages 25-29; it stands second to the United States at younger ages and second to Netherlands at older ages.

MARITAL FERTILITY RATES

The fertility rates shown up to this point are based upon births during given calendar years as they have related to total populations and to total female populations of given age regardless of marriage.

Another conventional type of fertility rate is one based upon total number of *children ever born* per married woman of given age. Whereas the annual fertility rates are usually based upon the registrations of vital statistics, the cumulative fertility rates are usually derived from census inquiries on number of children ever born.

Although Ireland is characterized by conspicuously low proportions married and by late age at marriage, her marital fertility rates are conspicuously high. This is brought out in Figure 7 showing number of children ever born per 100 marriages existing at census dates (*circa* 1946-1950), by duration of marriage. The rates are conspicuously higher in Ireland than any other country represented for women married five years or more. Netherlands stands in second place. The rates for Australia and the United States are in middling position, and those for Switzerland, France, and Great Britain are relatively low.

Caution must be used in comparing Figures 6 and 7 because of differences in type of rate. There is the strong suggestion, however, that (a) the excess of the general fertility rate for women 15-24 years old in the United States over that for Ireland is due entirely to higher proportions married, (b) the excess of the same United States rate over that for England and Wales arises from higher proportions married and higher marital fertility and (c) the relatively low general fertility rates of United States women 35-44 years are due to low marital fertility rates in the United States at these ages.

United Nations data on cumulative fertility rates for married women in several countries, by duration of marriage and age of wife at marriage, points, up the inherently high fertility of the women of Ireland who do marry at young ages (15, p. 65).

MARITAL FERTILITY IN THE UNITED STATES SINCE 1910

The United States Census data on number of children ever born reveal some interesting aspects of fertility trends since 1910. In Figure 8 the conventional age-specific cumulative fertility rates for all women and for ever-married women are shown for 1910, 1940, 1950, and 1957. Several points stand out:

1. Among the ever-married women the rates for 1910 are highest at all ages and those for 1940 are lowest at ages

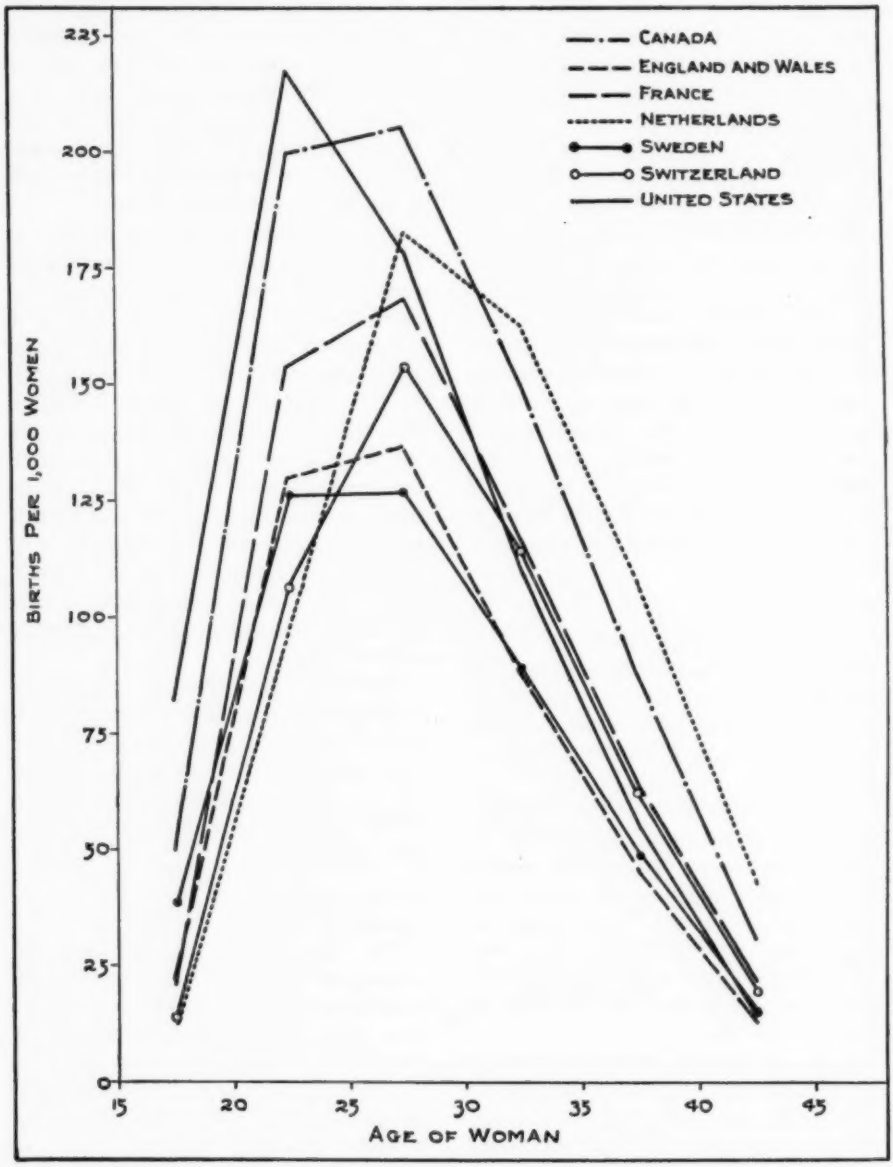

FIGURE 6. Age-specific General Fertility Rates. Average annual number of live births per 1,000 women of specified age in selected countries for the period 1950-1954. (Reference 15, pp. 134-135.)

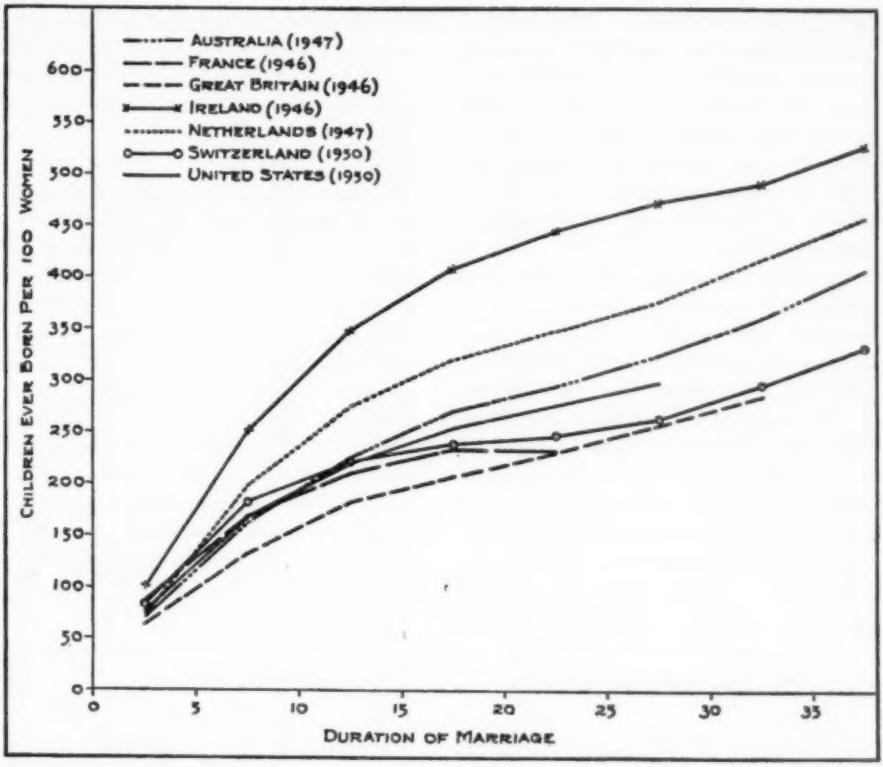

FIGURE 7. Number of children ever born per 100 marriages existing at specified census date, by duration of marriage in years, for selected countries. (Reference 15, p. 62.)

under 35. However, the rates were almost as high in 1957 as in 1910 for women under 25 years old.

2. The general fertility rates, those for all marital classes, were actually higher in 1957 than in 1910 for women under 30 years old.

3. The 1950 rates were higher than the 1940 rates for women under 35 years old but were lower for women of older ages.

4. The 1957 rates surpassed those of 1950 at all ages except the oldest (45-49).

That the rates for women 40-44 and 45-49 years old were lower in 1950 than in 1940 simply means that women 40 years of age and over in 1950 were too old to be affected much by the baby boom of the preceding decade. They still reflected the long-time downward trend in size of completed family. However, the women who will be 40-44 years old in 1960 will be those who were affected by the baby boom. During the decade of the sixties, therefore, we may expect a reversal of the long-time downward trend in size of completed family. This is also pointed up by comparison of the fertility rates of women of specific

ages in the same Census. Thus, in 1957 the fertility of women 35-39 years old actually surpassed that of women 40-44 years old, and the average fertility rate for the latter group, in turn, was higher than that for women 45-49 years old. This is a clear demonstration of increase in average size of completed family.

Figure 9, based upon the same data, shows the percentage of women of specified age group bearing specified numbers of children in 1910, 1940, 1950, and 1957. This throws considerable light on the source of the increases in fertility since 1940. There were marked *decreases* in the proportion of women bearing no child and bearing only one child. On the other hand, there were marked *increases* in the proportions bearing two and three children. The increase in average number of children has been due not to a return of the very large family of the 1910 era but to increases in proportions bearing two and three children.

COHORT FERTILITY

In assaying fertility trends, it is important to keep in mind the difference between "period" and "cohort" fertility. Figures 8 and 9 relate to period fertility, i.e., average number of children ever born among women of specific ages at specific periods.

Figure 10 adds another dimension and charts the fertility histories of specific cohorts of ever-married women, i.e., women born in a given period. The term "cohort" simply implies that the women are "followed through" for observation regarding events of pertinence such as their fertility. Figure 10, based

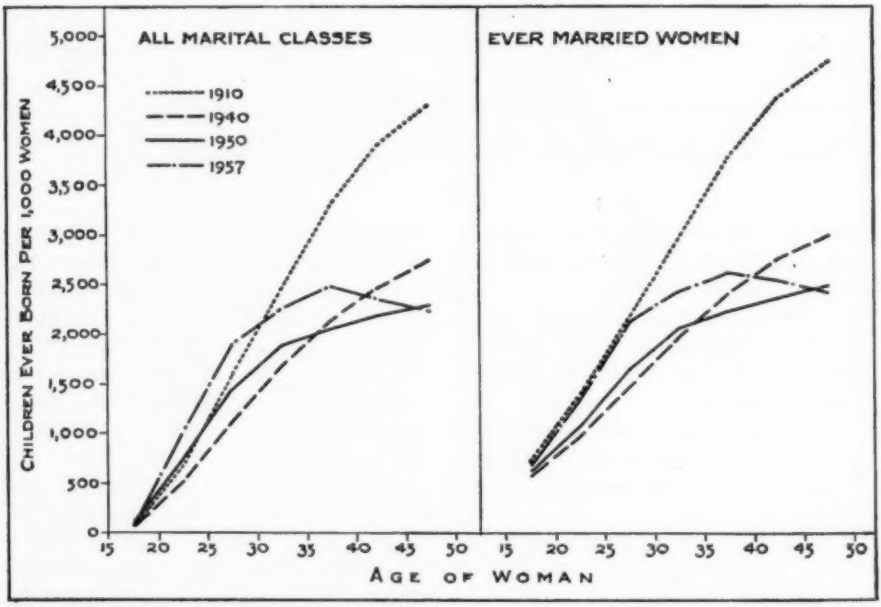

FIGURE 8. Age-specific general and marital fertility rates in the United States, 1910, 1940, 1950, and 1957. (References 8, 9, 10, 11.)

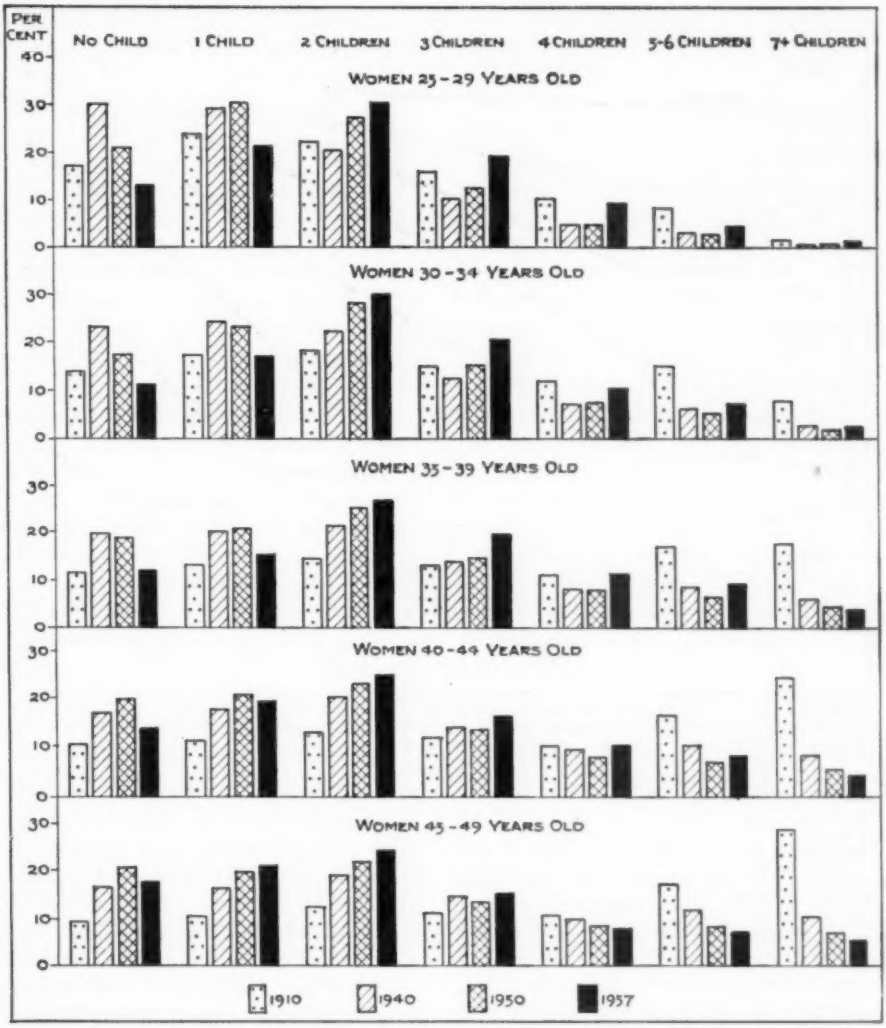

FIGURE 9. Percentages of ever-married women of given age reporting specified number of children ever born. United States, 1910, 1940, 1950, and 1957. (References 8, 9, 10, 11.)

upon Current Population Survey data for 1957, gives the fertility of women of specific birth cohorts in given calendar years (11). Thus the top line is that for women born during 1895-1900 and, hence, 40-44 years old in 1940. The lines below are for women born in successive five-year periods and, hence, successively younger.

The higher fertility of the younger cohorts by given ages may be visualized by comparison of similar symbols on the

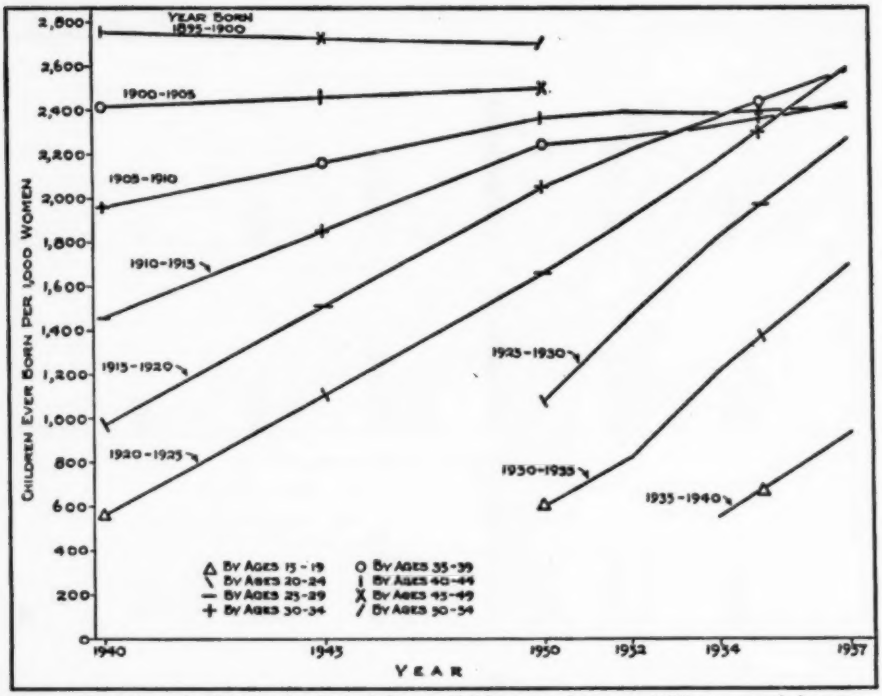

FIGURE 10. Cohort fertility in the United States. Children ever born per 1,000 ever-married women, for specified five-year birth cohorts, by year of enumeration and age at given dates. United States, 1940, 1950, 1952, 1954, and 1957. (Reference 11, p. 9.)

cohort lines in Figure 10. Thus by noting the small circles, we see that the low point in the downward trend in the *average* number of children ever born by ages 35-39 was reached by women born during 1905-1910, i.e., it occurred around 1945; more recent cohorts have exhibited increases. The low point in fertility by age 30-34 was reached by women born during 1910-1915. This occurred around 1945.

DIFFERENTIALS BY COLOR, RELIGION AND RESIDENCE

There has been a remarkable increase since 1940 in the fertility of young nonwhite married couples in urban areas of the United States. In 1940, the urban nonwhite married women in this country were characterized by lower average fertility than urban whites of similar age. This was due almost entirely to higher proportions childless. The fertility of *mothers* tended to be higher among nonwhites than whites. Similar differences by color still obtained in 1950 among women 25 years of age and over. Among the urban married women under 25 years old, however, the nonwhites surpassed the whites of comparable residence with respect to average number of children and fell below the whites with respect to proportions childless. This increase in fertility of young nonwhites has been attributed, in part, to a cleanup of venereal diseases since 1940. Other factors

are the general improvement in the economic, social and civic status of nonwhites in this country since 1940 (4).

Regional differences in fertility still exist in the United States as in other modern countries. In general the more industrialized and the more urbanized the area, the lower the fertility rates tend to be. In the United States the fertility rates tend to be relatively low in the Northeast and Pacific areas, intermediate in the North Central areas, and high in the South and Mountain areas. The high fertility of the South is due partly to the high proportion of nonwhites as well as to the relatively high proportions rural in the population.

The tendency for fertility to be lowest for urban people, intermediate for rural-nonfarm people, and highest for the rural-farm still persists but not nearly so sharply as in previous times. Not only is the urban-rural differential in fertility narrowing, but the proportion of people classified as rural is decreasing. It was 60 per cent in 1900 and 41 per cent by comparable definition in 1950. In 1910, the farm population contributed about one-third of the total in the United States. In 1955, it comprised less than 14 per cent. There has been a sharp increase in proportion of people living in large metropolitan areas. In 1950, slightly over one-fourth of the people of this country lived in the twelve urbanized areas of one million or more population (7).

With regard to the three major religious groups in the Western countries, fertility rates tend to be highest among the Catholics, intermediate among the Protestants, and lowest among the Jews. For instance, the average number of children ever born per 1,000 ever-married women 45 years old and over in the United States in 1957 was 3,056 for the Catholics, 2,753 for the Protestants, and 2,218 for the Jews (10, p. 41).

Some of the mating and fertility characteristics may be summarized for specific countries. As indicated in Table 1, the United States is characterized by highest marriage rates, youngest age at marriage, highest proportion ever married among women under 25 years old, and youngest age structure of the ever-married women among the countries considered. Largely because of the high proportions married, women 15-19 and 20-24 outrank those of the other countries considered in general fertility rates. However, the fertility rates for women 30 years of age and over are relatively low.

Because of the relatively high fertility at young ages and relatively low fertility at older ages the proportion of annual births attributable to women under 25 is higher for the United States than for the other countries considered. In 1954 the proportion was 46 per cent for the United States as compared with 19 per cent for the Netherlands and possibly an even lower proportion for Ireland. In the remaining countries the percentages ranged from 26 to 38 per cent.

The Netherlands also has relatively high crude marriage and birth rates but a relatively late age at marriage. Therefore among countries represented in Figures 5 and 6, which incidentally do not include Ireland, the Netherlands is characterized by lowest general fertility rates at young ages and highest at older ages.

Canada is distinctive for her highest crude birth rates during the past decade. She has a relatively high marriage rate and stands next to the United States with respect to high proportions married

TABLE 1

RECENT INDICES OF MARRIAGE AND FERTILITY FOR SELECTED COUNTRIES

Country	Crude Marriage Rate (1957) [1]	Crude Birth Rate (1957) [1]	Median Age at First Marriage Circa (1950) [2]	Per Cent			General Fertility Rate		Cumulative Marital Fertility Rate by Duration of Marriage (1950-51) [4]	
				Ever Married Among Women 15-24 (1950-51) [3]	Ever Married Women 15-49 Who are 15-24 (1950-51) [3]	Births Occurring to Women 15-24 (1954) [4]	Women 20-24 (1954) [4]	Women 30-34 (1954) [4]	5-9 Yrs.	15-19 Yrs.
Australia	7.7	22.9	22.0[b]	27.7[b]	12.6[b]	37.1	198	122	164[b]	271[b]
Canada	8.2	28.6	21.7	30.2	13.8	35.6	220	160	—	—
England and Wales	7.9[a]	15.6[a]	22.0	27.3	10.2	36.0	136	85	134	209
Finland	7.2	19.8	22.9	23.2	11.1	31.6	154	123	210	320
France	7.0	18.4	21.9	24.4[c]	11.5[c]	32.7	156	110	169[a]	231[c]
Ireland	5.0	21.2	26.0	8.7	5.9	—	—	—	251	409
Netherlands	8.5	21.2	24.3	17.0	9.2	18.7	99	162	200	322
Norway	7.1	18.2	23.5	19.6	8.0	30.1	138	117	174	240
Sweden	7.0	14.6	22.8	23.0	8.5	37.5	125	86	—	—
Switzerland	8.1	17.7	24.5	14.3	6.8	26.1	105	110	182	237
United States	8.9	25.0	20.2	43.6	16.4	46.1	236	116	168	253

[a] Year 1956 [b] Year 1947 [c] Year 1946

[1] *Population Index*, July, 1958, Vol. 24, No. 3, pp. 298-305.

[2] For explanation and source, see Figure 2.

[3] DEMOGRAPHIC YEARBOOK, United Nations, 1955, Table 12, pp. 390-429.

[4] United Nations: *Recent Trends in Fertility in Industrialized Countries*, New York, 1958, pp. 23, 60, 135, 137.

among women 20-24 years old. She stands highest in general fertility rates at ages 25-29. By standing next to the United States in high fertility at young ages and next to Netherlands with respect to high fertility at older ages, Canada is distinctive in her exhibition of relatively high fertility at all ages.

Ireland is characterized by lowest marriage rate, oldest ages at marriage, lowest proportions married among women under 25. On the other hand, the fertility rates of the women who do get married probably outrank those of any other country in the Western world. Hence her crude birth rate and general fertility rates tend to be in middling position despite the disadvantage of late age at marriage.

England and Wales and Sweden, in contrast to Canada, exhibit relatively low fertility levels at all ages. They are in middling position with respect to age at marriage. The remaining countries tend to fall in middling positions with respect to most characteristics considered but some of them also display certain distinctive features. It may also be noted that whereas the relatively high fertility of several countries might truly be ascribed partially to the influence of given religions or ethnic groups it is also apparent that the fertility patterns of Catholics in Ireland, for instance, are tremendously different from those of France.

DEMOGRAPHIC SIGNIFICANCE

The chief demographic consequence of the mating and fertility patterns has been the large increase in population. The increase in population during the past two decades has been a world-wide trend. However, in underdeveloped countries the increase has stemmed largely from decreases in mortality. In the

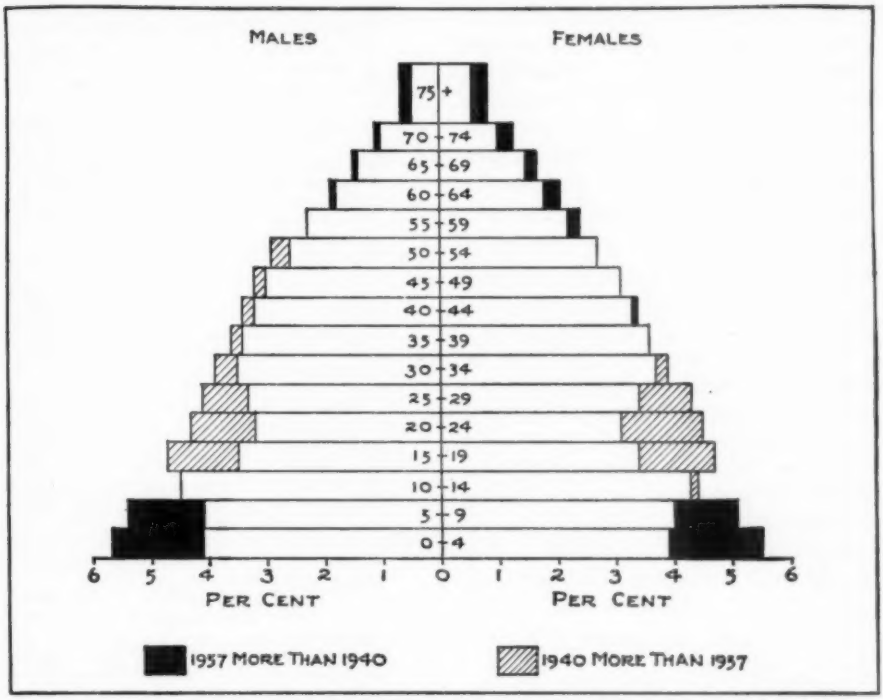

FIGURE 11. Age pyramids for the United States, 1940 and 1957. (Reference 10, pp. 25 and 27.)

modernized countries the recent increase in population has stemmed mainly from increases in fertility.

A second demographic consequence is that of change in age composition. Since increased births were the source of the increased population in the modernized countries the increase has been conspicuous in the young age group. In 1940 there were 45.3 million people under 20 years old in the United States. In 1957, the number was about 64 million. This was an increase of about 40 per cent. At the same time the population was becoming older. The number of persons 60 years of age and over increased by 58 per cent from about 13.7 million to 21.7 million. This increase in the number and proportion of aged persons and the implications for future prevalence of mental illness and institutional care and treatment are perhaps of special concern to the mental hygienist. The general change in age composition in the United States is shown by the age pyramids for 1940 and 1957 (Figure 11).

The demographic consequences could be discussed at further length. The changing geographic and urban-rural distribution, and the burgeoning of suburbs and huge housing developments, are not unrelated to the postwar increases in size, age structure, and to marriage and reproduction trends.

The previously mentioned narrowing of urban-rural differentials in fertility

is but one aspect of a general disappearance of urban-rural contrasts as we once knew them. With the coming of the radio and television, the surfacing of roads, the popularization of the automobile, the increasing tendency for rural people to go to the city to work and for the urban people to come to rural areas to live, the urban and rural people and the people in different regions and classes are becoming more alike in many ways other than their fertility behavior (3).

However, whereas there is a trend toward uniformity of fertility patterns within the United States and probably also within other countries, we have also noted that the modern Western countries present a variety of patterns of mating and fertility. Thus if I was supposed to describe a norm or benchmark, I have failed to deliver. The norms and benchmarks must be found in the specific countries. More specifically, if one desires to study the relation of fertility to given psychiatric conditions, he will do well to follow the time-tested method of using as a control group people that resemble the subjects as much as possible in all things except the existence of the psychiatric condition.

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DISCUSSION

ROBERT C. COOK*

The Galtonian concept of eugenics envisioned the enhancement of the inborn quality of future generations by the manipulation of forces under social control.

Some form of selection is the only way in which such an enhancement of inborn qualities can be brought about.

"Natural selection" has been accomplished mainly through differential survival. Artificial selection involves selective mating rather than selective survival. The breeder chooses the parents arbitrarily; the eugenist hopes social forces will do a reasonably effective job of facilitating favorable choices.

In the context of such a background, Dr. Kiser's analysis shows that in human cultures it is unquestionably possible to develop social forces which could be effective to increase the proportion of individuals of high genetic potential. The Irish pattern, where only about one female in five is married during the years of her maximum fertility, is in sharp contrast to the United States pattern where three-quarters of the females are married during the period of maximum fertility.

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In the United States, with a fertility pattern approaching pangamy and a low death rate, one can hazard the guess that the eugenic prognosis is not very favorable. In addition, the existence of a pattern of differential fertility which would appear to be dysgenic rather than eugenic would not seem to enhance the eugenic prognosis.

If such considerations have any validity, Dr. Kiser's paper would appear to offer reassurance that fertility patterns in a societal context do exist which might be eugenic if birth selection is favorable. Perhaps what is needed is a human biological analogue of Maxwell's demons who in effect herded the molecules in their random wanderings. Without any effective steps to develop social patterns which will constitute positive and reliable mechanisms for promoting adaptive birth selection, eugenics remains a paper discipline. Until this is done it will continue to be a matter which is solemnly discussed; but like Charles Dudley Warner's weather, it will remain a subject about which nothing effective is being actually done.

DIFFERENTIATING EFFECT OF INTELLIGENCE AND SOCIAL STATUS

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EVEN PRIOR TO THE ANALYSIS of intelligence test scores in relation to family size, data were available which suggested an association between intellectual level and fertility. An individual's occupational and educational level reflect in large part his effectiveness in meeting a long succession of intellectual demands. Intelligence tests provide a more objective measure of a relatively standardized and limited sample of intellectual behavior. On the other hand, the behavior samples underlying occupational and educational level have the advantage of broader coverage and longer duration. Socioeconomic variables, educational level, and intelligence tests may thus be regarded as separate indices of intelligence. As might be expected, these indices are highly intercorrelated among themselves. In relatively unselected adult samples, for example, intelligence test scores correlate over .70 with amount of schooling (Anastasi, 1958, pp. 205-206). Such correlations are about as high as those among a number of different intelligence tests.

SOCIOECONOMIC AND EDUCATIONAL VARIABLES

Differential fertility in various subgroups of the population was recorded as early as the seventeenth century in Europe (Kuczynski, 1935). Sufficient data were subsequently gathered in the United States and in several European countries to establish a significant inverse relation between fertility and such variables as income, occupational level, and amount of education (Kiser, 1942; Lorimer and Osborn, 1934; Sewell, 1944; United Nations, 1953). The widespread decline in the birth rate which began in the nineteenth century was more rapid in the upper than in the lower socioeconomic and educational classes, thus either producing or augmenting these fertility differentials. One of the most persistent differentials, occurring even when other subgroup differences in family size are absent or reversed, is that between urban and rural groups. This difference usually increases when comparisons are made between agricultural and non-agricultural occupations. The largest families are thus found among those rural residents who are engaged in agriculture (United Nations, 1953).

These well-established relationships between fertility and socio-educational variables must nevertheless be qualified in at least three ways. First, at the highest socioeconomic levels, the negative correlation between family size and income tends to disappear or even to be reversed. Evidence on this point is provided by surveys conducted in several American and European cities (Kiser and Whelpton, 1944, 1958; United Nations, 1953; Whelpton and Kiser, 1946-1955). One hypothesis proposed to account for such findings is based upon the

possible role of upward social mobility in restricting family size (Westoff, 1953). Some indirect evidence for this hypothesis was provided by the finding that Philadelphians listed in both the 1940 *Who's Who in America* and the *Social Register* had larger families than those listed only in *Who's Who in America* (Baltzell, 1953). It was argued that the latter had more often achieved eminence through their own efforts rather than having enjoyed social prominence throughout their lives. They had thus shown more upward social mobility than the *Social Register* subgroup. Other analyses conducted within the same study, involving persons with a private school education and those whose families had been listed in *Who's Who* since 1900, likewise suggested greater fertility among the socially less mobile groups. A more recent survey of a random sample of men listed in the 1956-57 edition of *Who's Who* corroborates some of these earlier findings (Kirk, 1957). Attempts to test the social mobility hypothesis among intermediate socioeconomic groups, however, have yielded inconsistent results (Berent, 1952; Kiser and Whelpton, 1958). At such levels, it is difficult to identify persons for whom the drive for upward social mobility can be assumed to be negligible, even when objective evidence for such mobility is lacking.

A second major exception to the inverse relation between socioeconomic level and fertility is to be found in high-fertility areas, where the crude birth rate is approximately 40 or more per thousand. Although demographic data from such areas are generally meager, when available they often reveal an absence of the expected fertility differentials. In Brazil, for example, socioeconomic differentials are slight and not in the expected direction. Thus within each type of work, the fertility of employers and own-account workers tends to be higher than that of employees and unpaid family workers (United Nations, 1953).

The third and most important point pertains to the narrowing of fertility differentials following the postwar rise in birth rate. Surveys conducted both in the United States (U.S. Census Bureau, 1948, 1953) and in several European countries (Carter, 1953; United Nations, 1953) indicate that fertility increased more among those classes in which the prewar level had been lower, that is, among urban residents and persons in higher educational, vocational, and income levels. Some of the findings in a 1952 sample survey conducted by the U. S. Census Bureau illustrate these changes (U.S. Census Bureau, 1953). Thus income level showed no relation to number of children, except for a proportionately larger number of children at the lowest income level (under $1000). Moreover, professional, semi-professional, and managerial groups had proportionately more children than did certain lower occupational groups, such as clerical and sales workers. Fertility differentials among educational levels had likewise decreased (U.S. Census Bureau, 1948), although the remaining differences among educational groups were still large, as were those between urban and rural dwellers. Also relevant are recent surveys of certain highly selected groups, including men listed in *Who's Who in America* (Kirk, 1957) and former scholarship holders among the graduates of a British grammar school (Hutton, 1953). In both groups, the number of offspring suggests that the current replacement rate may be even higher than that of the general population.

It should be added, however, that the most recent sample surveys conducted by the U. S. Census Bureau indicate that the above qualification regarding fertility differentials must itself be qualified (U.S. Census Bureau, 1958). Although from 1950 to 1957 the over-all birth rate continued to rise, fertility differentials with regard to educational, occupational, and income level again *widened*. At the same time, there occurred a *narrowing* of the urban-rural differential in fertility.

INTELLIGENCE TEST PERFORMANCE AND FAMILY SIZE

Turning now to investigations of the relation between intelligence test scores and family size, we must recognize at the outset that the problem is beset with methodological complications (Anastasi, 1956). Many of these difficulties center around sampling. Let me cite a few by way of illustration. Except for a few studies which will be separately reported, the relationship investigated was that between child's IQ and number of siblings, rather than that between parental intelligence and number of offspring. Such an approach fails to take into account the relative proportion of childless families and unmarried persons at different intellectual levels; nor does it consider differential mortality rates. Size of sibship is usually based on number of siblings alive at the time of the survey, rather than on total number of pregnancies, number of births, or number of children born alive. In two studies in which intelligence was correlated with both number of living sibs and total number of births in the same groups, the latter correlations were higher. Such a finding indicates that differential child mortality partly offsets differential fertility (Dawson, 1932; O'Hanlon, 1940).

Most studies have included a large number of incomplete families of relatively young parents. Children from such incomplete families are of course classified as belonging to smaller families than will eventually be the case, a procedure which will tend to underestimate the relation between intelligence and family size. On the other hand, a negative correlation between intelligence and family size in a sample of incomplete families could simply reflect a tendency for the less intelligent parents to begin having children earlier; it need not follow that they would have larger completed families. A further complication is that younger parents in general are likely to be better educated than older parents surveyed at any one time, owing to the rising educational level of the population.

The most extensive analysis of the relation between intelligence and family size is to be found in the 1947 Scottish Survey (Scottish Council for Research in Education, 1949, 1953, 1958). In that year, a group intelligence test including verbal and pictorial content was administered to a nearly complete sample of 11-year-old children in Scotland. The correlation between test score and size of sibship was —.28. Within a random sample of 1215 children individually examined with the Stanford-Binet, the corresponding correlation was —.32. The mean Stanford-Binet IQ dropped steadily from 113 for "only" children to 91 for children with five siblings. The tendency for children from larger families to score lower persisted within each occupational class, a finding also reported by other investigators. Since fairly broad occupational categories were employed, however, it cannot be assumed that socioeconomic differences have been ruled out within single occupational levels.

Another large-scale investigation was conducted among 6-to-12-year-old French school children during the years 1943 and 1944 (Gille *et al.*, 1954; Heuyer *et al.*, 1950). This survey included approximately a two per cent sample of the elementary school population of France, chosen so as to yield a representative geographical distribution. All subjects were given René Gille's "test Mosaïque," a pictorial group test of intelligence. Mean scores again exhibited a consistent drop with increasing family size. This differential was apparent within each year group, the difference in mean score between a one-child family and a family of eight or more corresponding to 1 or 2 years of mental age. Within occupational groups, the negative relationship between intelligence and family size was clearly apparent among farmers, manual laborers, and clerical workers; it was barely discernible in the managerial group and negligible in the professional class.

Other similar surveys have been conducted on a less extensive scale in several countries. In England, there have been a number of attempts to test complete age samples of children within limited areas, such as a city or small rural region (Bradford, 1925; Burt, 1946; Cattell, 1936, 1937; Roberts, 1939; Roberts *et al.*, 1938; Sutherland and Thomson, 1926). American investigations, although utilizing more heterogenous and less well-controlled samples, generally included adequate numbers of cases, ranging from several hundred to several thousand (Burks and Jones, 1935; Chapman and Wiggins, 1925; Damrin, 1949; Hirsch, 1930; Lentz, 1927). Those dealing largely with completed families report results closely similar to those of other studies. Mention may also be made of an investigation of Athens school children with a Greek adaptation of the Stanford-Binet (Papavassiliou, 1954) and of two studies of German school children in which grades, scholastic retardation, and other academic indices provided the data on intelligence (Eydt, 1938; Heinen, 1938). All of these studies confirm the findings of the more extensive surveys regarding the decline in mean intelligence test score with increasing size of sibship. When correlations were computed between test score and family size, they were uniformly negative and clustered in the .20's and low .30's.

Studies of certain special groups have yielded similar results. In a survey of over 20,000 retarded children in the Massachusetts public schools, mean IQ declined from 73.2 for "only" children to 68.9 for those with 9 or more sibs (Dayton, 1935). In the California Gifted Child Study, Terman and his associates found a correlation of —.27 between IQ and size of sibship within 91 completed families (Terman *et al.*, 1925). Although all families are now complete, information on number of sibs was not obtained in the later follow-ups of this group. Data on the relation between intelligence and number of offspring of the gifted subjects themselves, however, are available and will be analyzed and published in the near future.[1]

Of special interest are a few studies in which *parental intelligence* was correlated with number of offspring. An investigation on rural New England samples (Conrad and Jones, 1932) yielded low negative correlations between family size and parental intelligence test scores or educational level, only two of the correlations reaching significance. In completed families, moreover, all corre-

[1]Personal communication from Melita H. Oden, Stanford University, January, 1958.

lations were *positive* and none significant. Insignificant correlations were likewise found in a California study of completed families (Willoughby, 1928).

In a third investigation (Willoughby and Coogan, 1940), 12-year follow-up data were gathered on 373 graduates of four Providence, Rhode Island, high schools. No relationship was found between high school intelligence test scores and age at marriage, age at birth of first child, or number of offspring at the time of the survey. It is also interesting to note that a low but significant negative correlation was found between intelligence and number of siblings of the subjects themselves. Moreover, subsequent education and occupational levels of the subjects revealed an inverse relation to number of offspring. Although this study is inconclusive because of the relatively small and selected sample and because of the incompleteness of the families, it nevertheless has the merit of providing a measure of parental intelligence prior to educational and vocational differentiation.

A more extensive longitudinal survey involved a 20-year follow-up of 300 engaged couples originally living in New England (Mishler *et al.*, 1955; Westoff *et al.*, 1957, 1958). Initial age of the group averaged about 25 years. The correlation between initial Otis Intelligence Test scores and eventual number of live births in a subgroup of 145 couples was .24 for women and .27 for men. These cases represent all couples living and married at the time of the follow-up, on whom sufficient information was available and who met certain additional requirements for inclusion in this analysis (Westoff *et al.*, 1957, pp. 492-493). Both correlations are significant at the .01 level and indicate a slight tendency for the brighter parents to have *larger* families. It is noteworthy that none of the studies in which parental intelligence was correlated with number of offspring has so far yielded the negative correlation ordinarily found when child intelligence is correlated with size of sibship.

ANALYSIS OF CAUSAL RELATIONS

In explaining the negative correlation between intelligence and size of sibship, three different etiological mechanisms should be considered. The actual causal relations may, of course, involve any combination of two or all three of these mechanisms. First, there may be *inherited factors* (neural, glandular, biochemical, etc.) which serve as constraints, reducing adaptability of behavior and limiting the sort of intellectual development measured by current intelligence tests. The less able parents would thus transmit their hereditary limitations to their offspring. The obtained correlations would then result from the fact that, within a given culture or subculture, persons with inferior heredity tended to have more offspring.

A second explanation attributes individual differences in children's abilities to *psychological differences in the environments* provided by parents of varying intellectual levels. In this case, the correlation between family size and intelligence of offspring would again result from a tendency for the less intelligent parents to have more children, but heredity would not be involved. Differences in intellectual level among the parents, as well as among the offspring, would have resulted from environmental factors.

A third possible interpretation is based upon *family size* itself as a causal factor. A larger family, for example, usually means a reduction in per capita funds available for education, recreation,

housing, food, medical attention, and other environmental requisites. From a psychological viewpoint, a significant factor is the degree of adult contact provided in families of different sizes. Available evidence indicates that such contact may be one of the most important single factors in language development, which in turn plays a major part in both educational progress and intelligence test performance. Whatever the specific factors involved, the etiological mechanism under consideration is independent of the intellectual level of the parents.

On the basis of existing data, it is impossible to choose among these explanations or to determine the relative contribution of the three types of influences. Certain lines of investigation do nevertheless throw some light on causal relations. Among them may be mentioned the previously cited studies on parental intelligence and number of offspring, as well as research on the role of language in the relation between sibship size and intelligence (Nisbet, 1953a, 1953b; Scott and Nisbet, 1955) and studies on families artificially limited by death of the father (Sutherland, 1930). Taken together, these investigations suggest that no one hypothesis is sufficient to account for all the observed correlation between intelligence and family size. But further than that it would be hazardous to go.

EUGENIC IMPLICATIONS

Regardless of which causal mechanism is involved, a negative correlation between intelligence and family size could lead to a decline in intelligence in successive generations. Between 1930 and 1950, a number of demographic writers predicted such a decline, with varying degrees of foreboding. Subsequent follow-ups, however, have revealed a rise in mean intelligence, rather than the predicted decline. Evidence for such a rise is to be found in the 1932 and 1947 surveys of 11-year-old Scottish children (Scottish Council for Research in Education, 1949), in a comparison of the intelligence test performance of the United States Army in the two World Wars (Tuddenham, 1948), and in an analysis of the test scores of American high school students over a twenty-year period (Finch, 1946). Other studies conducted on a smaller scale obtained essentially corroborative results (cf. Anastasi, 1958, pp. 209-211).

One explanation offered for the observed rise in intelligence test performance is the increasing test sophistication of the population, which would effect an improvement in test-taking abilities with no accompanying rise in general level of intellectual functioning. In the Scottish Survey, it was estimated that no more than 25 per cent of the rise in score could be attributed to this source (Scottish Council for Research in Education, 1958, pp. 39-47). Improvement in educational and other cultural facilities, together with the rising educational level of the general population, represents a second type of influence, of much broader scope than test sophistication. From a purely genetic point of view, it has been demonstrated that a low negative correlation between intelligence and family size is not inconsistent with a rising intellectual level (Penrose, 1948, 1950a, 1950b; Willoughby and Goodrie, 1927). This follows from such conditions as the infertility of low-grade mental defectives, assortative mating, and the size of the parent-child correlation in intelligence. In conclusion, let me emphasize that whether we wish to establish the exact relation between intelligence and fertility, analyze its causes, or

consider its eugenic implications, we are faced with highly intricate questions to which there are no simple answers.

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DISCUSSION

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In view of the international scope of this conference, the following quotation from *Punch* might not be inappropriate to introduce a discussion of the differentiating effect of intelligence and social status on fertility trends:

> The figure of 2.2 children per adult female was felt to be in some respects absurd, and a Royal Commission suggested that the middle classes be paid money to increase the average to a rounder and more convenient number.

Whether differential support for the propagation of some members of society as opposed to others creates the problem we are discussing or whether this problem arises because nature is profligate in the propagation of some organisms and chary with others, is difficult to determine. But it is

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certainly true that bounties for every additional child offered in some states to families on relief may account for some of the differential fertility observed.

In the search for a possible connection between size of family and intelligence of offspring, the factor of socioeconomic status comes to mind. Letting intelligence be represented by X_1, size of family by X_2 and socioeconomic status by X_3, we can ask what the correlation between X_1 and X_2 would be if X_3 were held constant. Now r_{12} is generally found to be between — .20 and — .30, say — .25 on the average; r_{13}=ca .50. If $r_{12.3}$ (the partial correlation between intelligence and size of family when socioeconomic status is held constant) is to vanish, then

$$r_{12.3}=\frac{r_{12}-r_{13}\,r_{23}}{\sqrt{1-r^2_{13}}\ \sqrt{1-r^2_{23}}}=0$$

$$\text{or } r_{23}=\frac{r_{12}}{r_{13}}=\frac{-.25}{.50}=-.50$$

Hence, the correlation to r_{23}, size of family and scioeconomic status, must be — .50, if the socioeconomic factor is the connecting link between size of family and intelligence. The correlations reported in the literature run a little lower (in the — .30's and — .40's), but are not too far removed from — .50 to vitiate the assumption that socioeconomic factors may account in large measure for the apparent association between intelligence of offspring and size of family.

From Anastasi's account of the relation between socioeconomic status and fertility, it seems clear that there are contradictory trends among the various studies. I wonder whether the well-known fallacy of inferring correlation within groups from correlations between groups may not be at the bottom of some of the contradictions. Another possibility is that all the findings are true, but based on different time periods, different populations and different tendencies within the same population. We seem to be in the middle of a whirlpool of changes in family limitation, a situation in which contrary findings are the rule rather than the exception.

In dealing with the direct relation between performance on intelligence tests and family size, Dr. Anastasi has catalogued a large number of confounding variables to which I believe I can add a little more confusion. If the correlation between intelligence of parent and offspring is indeed about .50, and if the correlation between intelligence of parents and size of family is about .25, it is a little difficult to understand why the correlation between the intelligence of a member of a given sibship and its size is negative, even though this is quite possible mathematically. I wonder what would happen if all three of these factors were correlated on the same population. Would the negative trend persist?

Perhaps the negative trend is a consequence of a bias resulting from the selection of only one member of each sibship—the one caught in the testing program—whose intelligence is then correlated with the size of his sibship. If instead, we computed the average intelligence of the entire sibship and correlated that with the size of the sibship, I wonder whether the correlation would not be different.

There are some morsels of evidence favoring the tenability of each of the scientific models which were proposed by Dr. Anastasi. The first model, which invokes a hereditary basis for the nexus between intelligence of parent and size of family, has some support from animal studies in Oslo by Rasmussen. He found a negative correlation between strength of sexual drive and fecundity. If we regard weak sexual drives in the animal world as evidence of lesser ability, the above finding offers a biological basis for a negative correlation between size of family and parental intelligence.

The second model, which invokes an environmental explanation for the transmission of lower intelligence by inferior parents, leaves unanswered the question of why inferior parents should be more fertile. Is it lack of inhibition, lack of knowledge of birth control, or what?

The third model in which family size as an environmental factor (rather than heredity) bears the burden of the explanation, is quite tenable and also has some animal work in its support. Seitz has found that mother rats who are given a litter of 6 to care for, are more maternal and protective than mother rats who have to care for a litter of 12. Furthermore, the previously proposed partial correlation of zero between size of family and intelligence, when the influence of environment is removed, would also lend support to an environmental source for the observed relation.

Perhaps a more parsimonious model might be erected on the assumption that basic intelligence is determined by hereditary factors, but that its expression is determined by environmental factors. To quote a recent telecast: "Heredity deals the cards, environment plays the game." The environment can elicit intelligence or occlude it, but it cannot create it. From this point of view, the negative correlation between size of family and intelligence of its members would be explained either by deprivation in large families or by greater probability of expression of recessive or other genetic tendencies as the size of the sibship increases. This kind of model seems to me to include within its structure most of the findings reported thus far. It has the added advantage of providing new testable hypotheses for experiments which can increase our knowledge, even if the model itself is finally discarded.

A word about techniques used in this field. The Pearson product-moment correlation may not be the method of choice for representing the relations. Perhaps regression methods in which the intelligence of a member of a sibship is predicted from the size of the sibship (a discrete number not subject to errors of measurement) would be a better method. Intra-class correlation may offer a still better method. One wonders about the linearity and homoschedasticity of the correlation between intelligence of children and size of their sibships.

The physical factors possibly connected with the production of offspring at the end of the reproductive cycle in mothers of large families, as well as conditions of paranatal care in families of low socioeconomic status may be significant variables, as shown in the studies of Lillienfeld and Pasamanick.

The identity of the psychological tests used to determine intelligence and their factorial composition also need to be examined carefully. Certain tests may inadvertently penalize children or adults from lower socioeconomic levels. For example, group verbal tests sometimes diagnose as feebleminded individuals who appear quite normal on the Porteus Maze Test.

If these studies can not be done successfully in man, an estimate of the biological substrate, if any, of the relation between intelligence and fertility can be obtained from animal studies. With the development of techniques for measuring comparative intellectual status of animals, such studies should prove most enlightening.

All in all, this has been a most interesting paper to review, and the fact that it raised more questions than it answered is indicative of its merit.

BIOCHEMICAL FERTILITY REGULATION

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FERTILITY CONTROL IN EVOLUTION

IN THE EVOLUTION OF HIGHER vertebrates reduction of the reproduction rate may be recognized as a characteristic feature, with most physiologic adaptations that result in fertility control appearing in the female of the species (for review, see Witschi, 1956). An amphibian or teleost ovary may produce hundreds of thousands of mature ova during the reproductive era, while the human ovary gives rise to a mere few hundred. Although the principal adaptations contributing to this fertility regulation have been described at the morphologic level, the translation of these events into terms of biochemical phenomena remains undone. We know, for example, that the process of follicular atresia contributes enormously toward reducing the fertility potential of mammalian vertebrates and that a further limitation is achieved by the characteristic single egg ovulations of most primates. The human ovary contains an estimated five to ten million germ cells at birth. Through the years this number decreases until the menopause when the last ovocytes disappear. Considering the small number actually released as mature ova, it is apparent that an overwhelming majority—almost the entire five to ten million—end by degeneration within the ovary. This atretic degeneration and the strict limitation of ovulation are normal and well-regulated processes, but the biochemical mechanisms involved have not been characterized. Restriction of the reproductive rate by limiting the total germ cell number is encountered along many evolutionary lines in which population restriction has an apparent survival value. In man's effort to exert physiologic regulation of his fertility, he has been unable to invoke this direct and basic method so successful in nature; this goal is rather remote. Trying to develop methods of biochemical fertility regulation, we accept our endowment of potential gametes—eggs and sperm—and attempt to exert willful control of one of the many other potentially vulnerable links in the chain of reproductive events.

The reproductive process in the mammalian female may be considered as a sequence of steps, each depending on the successful completion of the preceding ones (Nelson, 1956). These may be listed as: (1) production and release of hypophysial gonadotrophic hormones; (2) stimulatory action of gonadotrophins upon the ovary; (3) ovulation and entrance of the ovum into the Fallopian tube; (4) fertilization; (5) early development of the zygote during passage through the Fallopian tube; (6) tubal exit of the zygote and blastocyst formation; (7) nidation of the blastocyst in the endometrium; (8) maintenance of placentation and embryogenesis. With

the acceleration of research interest in fertility control that has occurred in recent years, each of these events has been focused upon in the attempts to develop acceptable methods to regulate the reproductive process.

STEROID ANALOGUES—ORAL PROGESTINS

The most advanced work, with newly developed steroid compounds, is based upon the ability of these substances to suppress the production of pituitary gonadotrophic hormones—the first step in the reproductive sequence. Secondarily, this influence on the hormonal control of gonadal function results in the inhibition of ovulation.

It is a classical endocrinologic concept that the sex hormones, estrogens, androgens and progesterone, engage in a physiologic feed-back mechanism which controls the rate of production and release of hypophysial gonadotrophic hormones. To a greater or lesser extent, each of these steroids has the capacity to suppress the pituitary production of gonad-stimulating hormones which are required for the normal process of ovocyte maturation and ovulation. As a practical method to affect ovulation suppression, however, each steroid type presents serious disadvantages. Androgens would be contraindicated because of the virilization that would be likely to occur in many women receiving effective doses. Estrogens present a multitude of problems including the fear, real or fancied, that the administration of estrogenic substances for a prolonged period of time might initiate neoplastic processes in the female reproductive tract. Also, these hormones would tend to produce endometrial hyperplasia and, in many instances, breakthrough bleeding. Experimental evidence has indicated that the third type of steroid hormone, progesterone, is at best a weak suppressor of pituitary gonadotrophins. Particularly when administered orally, large doses would be required and 100 per cent effectiveness could not be expected. Historically, at about the time that investigations had suggested the utility of oral progesterone in combination with small amounts of estrogen, many newly synthesized steroid analogues became available which, inherently, appeared to have effects on the reproductive system similar to the proposed combination. Three of these substances, 17-α ethinyl estraenolone (Enovid), 17-α ethinyl 19-nortestosterone (Norlutin) and 17-α acetoxyprogesterone (Prodox) are now being evaluated clinically for their effectiveness in the control of fertility.

The impetus for the development of these steroids did not come from a visionary view of their potential utility in oral contraception. Rather, the incentive behind their development was the need for cancer chemotherapeutics, for anabolic agents in the field of geriatrics, and, paradoxically, for the application of their activity in the treatment of some types of female sterility and habitual spontaneous abortion. Rock *et al.* (1956, 1957) observed that relatively low doses of 17-α ethinyl estraenolone had the dual action of suppressing pituitary gonadotrophins and maintaining a progestational type of endometrium. Utilizing these biologic effects, he was able to simulate menstrual cycles in women with long histories of amennorhea and lack of ovulation caused by insufficient pituitary gonadotrophin production. The compound was administered cyclically, about 20 days each month. During this time the hypofunctional anterior pituitary was completely suppressed and the endometrium developed to the progestational condition. Upon the with-

drawal of therapy, endometrial breakdown with menstrual bleeding ensued and shortly after the cessation of flow, the induced cycle could be repeated. Many women continued to menstruate regularly, *without therapy* after several months of artificially induced cycles. Frequently, the post-therapy cycles proved to be completely normal, with ovulation occurring. Presumably, the suppressed pituitary "rebounds" after the treatment, producing adequate gonadotrophins to initiate normal cycles. This experience in the field of infertility therapy made it apparent that steroid-induced pituitary suppression could be achieved without the untoward contraindications of the usual, steroid sex hormones. The subsequent contraceptive trials of the new steroid analogues have demonstrated the physiologic effectiveness of this mode of action for the biochemical regulation of fertility (Table 1) (Pincus *et al.*, 1958; Tyler and Olson, 1959). The extent to which this method will become acceptable for widespread usage will depend on considerations of cost, complexity of dosage, and experience with long-term drug administration. Regardless of eventual application, however, the method deserves recognition as an outstanding achievement toward controlling fertility by physiologic means.

TABLE 1

USE OF ENOVID AS AN ORAL CONTRACEPTIVE

No. of tablets missed•	*No. of cycles*	*Preg. rate/100 woman years*
0	1279	0.0
1- 5	282	9.2
5-19	151	25.9

Pre-treatment pregnancy incidence—62.5 ± 1.35

•Treatment involves app. 20 tablets of 10 mg. per month.
These data taken from Pincus *et al.*, 1958, describing their study in San Juan, Puerto Rico.

PLANT EXTRACTS—
LITHOSPERMUM RUDERALE

Animal experimentation with extracts of the North American prairie plant, Lithospermum ruderale, has provided evidence that gonadotrophic hormones, after being released by the pituitary, may be inactivated in the general circulation, and thus prevented from exerting their stimulatory influence on the gonad (Noble *et al.*, 1954). The work suggests that this material possesses an active constituent capable of influencing the gonadotrophin molecule in a manner that causes biochemical inactivation. An activity of this sort may offer an important advantage in avoiding an intrinsic interaction with the endocrine glands, for this is likely to minimize the possibility of undesirable, irreversible effects with long-term usage. Through the years, efforts to isolate a stable, active constituent from Lithospermum have not been successful. Breneman and Carmack (1958) have prepared spray-dried powders which have retained activity for as long as two years, but these investigators do not yet know the chemical identity of the active constituent. Gorman *et al.* (1956) have described extraction procedures for Lithospermum which have produced a crystalline material and several amorphous fractions, but more investigations are needed to determine if a specific component is related to the biologic activity.

IMMUNOLOGIC PHENOMENA

Among the intermediate steps between ovum maturation and zygotic development, the process of fertilization would present many advantages as a target for potential anti-fertility substances. At the present time, immunologic methods offer most promise in this approach. Establishment of the role of agglutinat-

ing substances (fertilizins) in the normal process of mammalian gamete fusion (Bishop and Tyler, 1956) has followed many years of exploration of this subject in invertebrate marine species. Tyler (1949) concludes that fertilizin and antifertilizin, when present on the surface of the respective gametes, facilitate fertilization. When present in solution, they block fertilization. If it is firmly established that gametes of the human produce interacting substances of this nature, it would be feasible to attempt controlled interference with this biochemical mechanism in the fertilization process. Lindahl (1954) has reported that mammalian spermatozoa are prevented from agglutinating, and, therefore, from losing their fertilizing capacity, because of the presence of a protein substance present in the semen for which he has introduced the term *male antagglutin*. He has demonstrated that antibodies can be formed which precipitate with antagglutins (Lindahl and Edlund, 1955). This type of investigation seems to hold great promise for the future. Another immunologic approach involves the formation of antibodies against the germ cells themselves; until now most work has been with the male gametes. Freund and his collaborators (1953) have been able to induce the loss of all germinal tissue from the testes of guinea pigs immunized with homologous testicular extracts. This has been confirmed by Katsh (1958) who, in agreement with Isojima *et al.* (1959), has found that extracts of guinea pig testis also induce sterility when used to immunize female guinea pigs. The mechanism of this latter phenomenon is not yet fully understood. One of the possibilities suggested has been that the immunized female is "protected" against spermatozoa, so that adequate quantities of antibodies appear in the female reproductive tract and inactivate spermatozoa deposited during coitus.

ANTI-ZYGOTE ACTIVITY

Until recently, the process of tubal passage and early cleavage of newly fertilized eggs was not seriously regarded as a likely vulnerable point in the reproductive process. With the analysis of the synthetic compound 1-(p-2-diethylaminoethoxyphenyl) l-phenyl-2-p-anisylethanol), referred to as MER-25, we have come to recognize this step as a likely site and time of action for potential antifertility compounds. On the basis of laboratory work it was established that MER-25 causes rapid degeneration of tubal ova (Segal and Nelson, 1958). Postulating that the activity in the rat would carry over to the human, it would offer an intriguing method of fertility control. Assuming the most ideal outcome of clinical tests, on the basis of what was seen in the rat, one might predict that the compound could be taken orally for a day or two subsequent to coitus and would prevent ovum development when and if fertilization occurred. Since implantation would never take place, there would be no disruption of the natural menstrual cycles and a fertile cycle would pass completely unnoticed. In a sense it would be like inducing pharmacologically in all cases what gynecologists believe occurs normally in 10 per cent or more of human fertilizations, i.e., the degradation of an ovum which, though fertilized, does not remain viable for the 8 days necessary to start the nidation process.

In the experimental work, post-copulatory events in female rats were studied following the administration of MER-25. The compound was given orally, in most instances at a dose of 25 mg/kilogram

body weight (Table 2). When treatment was initiated on the day of mating

TABLE 2

ORAL ADMINISTRATION OF MER-25
25 mg. per kilogram daily

Treatment days post-coitus	*No. of animals*	*No. of pregnancies*
Control	15	15
1- 4	20	0
2- 5	8	0
3- 6	8	0
4- 7	8	0
5- 8	8	8
5-21	4	4

and continued throughout the normal period of tubal passage of the ova, implantation never occurred. Treatment of similar length (4 days), including any part of the period of tubal passage, was equally effective in preventing pregnancies. When the 4-day treatment was initiated after tubal exit of the ova (days 5-8), not a single pregnancy was terminated and the fetuses developed normally. In fact, it has been possible to administer the compound continuously following the period of oviducal passage without impeding gestation, causing fetal resorption, or reducing litter-size. A single daily ingestion of 25 mg/kilogram body weight failed frequently to provide absolute protection against implantation and gestation, but when the dosage was raised from one-a-day to twice- and three-times daily, complete effectiveness was observed (Table 3). In fact, with 3-times-a-day administrations, dosage could be reduced to 8 mg/kilogram.

In studying the course of events within the Fallopian tubes, it was found that there is a blockage of early cleavages, so that ova of females treated with MER-25 undergo rapid degeneration shortly after fertilization.

TABLE 3

ORAL ADMINISTRATION OF MER-25
Single Day Treatment

Treatment day post-coitus	*Dosage mg/kilo*	*No. of animals*	*Full-term pregnancies*
1	25	8	6
1	25 b.i.d.	8	1
1	25 t.i.d.	15	0
1	8 t.i.d.	9	0
3	25	8	3
3	8 t.i.d.	14	0
5	25	8	8
5	25 t.i.d.	19	4*
5	8 t.i.d.	10	2**

*Additional 10 showed evidence of resorbed pregnancies
**Additional 2 showed evidence of resorbed pregnancies

From the studies in rats, it was not possible to determine if the early zygote deterioration resulted from an action directly on the egg, or on the Fallopian tube, altering it in such a manner that it no longer provided the necessary support for development of the egg. In order to test for possible direct action of the compound on ova, experiments were carried out with eggs of the American sea urchin, *Arbacia punctulata* (Segal and Tyler, 1958). This marine invertebrate presents several distinct advantages for a study of this nature. It provides an external fertilization situation, avoiding the complications of internal fertilization and viviparity. Several thousand eggs develop in synchrony, so that one does not have to depend on observations on isolated eggs. Also, development occurs very rapidly, so that deviations from normal can be observed in the living state, as they occur, under the scrutiny of the investigator.

The results indicate that minute concentrations of MER-25 have a direct blocking action on the fertilized eggs of the sea urchin. A concentration of 2×10^{-5} per cent added to the sea water medium five minutes after fertilization

blocks first cleavage of over 50 per cent of treated eggs. Those which initiate development do not progress beyond the blastula stage. At lower doses (2×10^{-7} per cent) the compound's inhibitory activity is manifest, also, although a small percentage of the zygotes may progress through gastrulation and develop into normal plutei. The blocking effect of MER-25 on fertilized eggs may be reversed by washing after exposure to even highly active concentrations.

The observations of the direct blocking effect of MER-25 on developing eggs has been extended to the mammalian egg by Chang (personal communication).[1] He has found that the isolated zygote of the rabbit, which can develop to the blastocyst stage *in vitro;* is prevented from doing so when small concentrations of MER-25 are added to the ambient medium.

With the realization that the compound acts directly on the egg, it became of interest to determine if pre-fertilization, follicular ova of mammals were susceptible to the biochemical influence of MER-25. Although the laboratory rodent is not the animal of choice to resolve this question, preliminary experiments have been carried out with the rat. Treatment was confined to variable periods of time before mating and the post-copulatory events were studied (Table 4). The results indicate a significant diminishment of fertility, which may have resulted from a deleterious influence on pre-ovulatory ova. Further investigations are in progress which may elucidate this matter.

[1]This information, to be published soon, was kindly provided by Dr. M. C. Chang, Worcester Foundation for Experimental Biology, Shrewsbury, Massachusetts.

TABLE 4

PRE-FERTILIZATION TREATMENT WITH MER-25 25 mg/kilogram per day, orally

Duration of treatment pre-coitus	No. of animals	No. of pregnancies	Average litter size
3-14 days	10	5	4.5
controls	12	10	9.5

ACTIVE CONSTITUENT OF PISUM SATIVUM (LINN)

From the studies of MER-25, mentioned above, it may be concluded that the compound is effective in preventing the nidation of a fertilized egg because of a direct blocking action on the egg itself. The investigations established that the endometrium of an MER-25 treated animal is capable of developing decidual tissue, necessary for placentation (Segal and Nelson, 1958). Another substance has been studied which is reported as having the same essential end result, i.e., the prevention of nidation, although the mode of action is entirely different. In the case of m-xylohydroquinone, the reported anti-placentation activity occurs as a result of an effect on the endometrium, vitiating its capacity to prepare for gestation. According to Sanyal (1956), the compound inhibits the peripheral action of progesterone, so that the endometrium does not develop adequately for nidation of the blastocyst. Although this activity in laboratory animals has not been confirmed by others (Thiersch, 1956a; Banik and Chakravarti, 1957), the substance has been given widespread clinical testing in India. Subjects are given oral tablets containing 350 mg. m-xylohydroquinone two times a month, on the 16th and 21st day. On the basis of the data now available, this method is said to have reduced fertility by 50 per cent under the conditions of the field trial.

More information is needed to determine if, indeed, the reported anti-progestational activity in animals can be demonstrated in human beings. This could be established readily by endometrial biopsies, performed following drug administration.

ABORTIFACIENT AGENTS—ANTI-METABOLITES

Subsequent to nidation and placentation, interruption of the reproductive process can be achieved by many substances which interfere with normal developmental processes. Thiersch (1956b) has studied a group of folic acid inhibitors (aminopterin) and amino acid analogues which are effective fertility inhibitors in animal tests. For example, diazo-acetyl-serine and diazo-oxo-norleucine, first isolated from cultures of streptomyces and later synthesized, were found to cause litter destruction in rats anytime from nidation to mid-term (Thiersch, 1957a,b). The mechanism is apparently a biologic antagonism, with the analogue blocking normal amino acid incorporation in protein synthesis. For eventual human application, compounds of this type would have to be administered under strict supervision. Suboptimal conditions of dosing could result in a partial effect without an actual termination of pregnancy, but interfering with the normalcy of embryonic development. Excessive doses could be harmful to the mother as well as the conceptus.

The studies that have been reviewed are relatively advanced toward development of acceptable measures for physiologic fertility regulation. No one method may be regarded as perfect; each has its limitations. Of equal, or perhaps greater importance, are the many studies being carried out with the hope of gaining a broader and more basic understanding of each link in the chain of reproductive events. Much remains to be learned about these processes before the ultimate opportunities for fertility regulation can be appreciated. It may be regarded as certain that many more leads will be developed from present laboratory investigations that will warrant evaluation as potential methods for the biochemical regulation of fertility.

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edited under the auspices of the National Research Council

EDITOR: CORRADO GINI

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INBREEDING EFFECTS ON MAN

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SOME FIFTY YEARS AGO, A. E. Garrod (1902) observed: "The question of the liability of children of consanguineous marriages to exhibit certain abnormalities, or to develop certain diseases has been much discussed, but seldom in a strictly scientific spirit. Those who have written on the subject have too often aimed at demonstrating the deleterious results of such unions on the one hand, or their harmlessness on the other, questions which do not here concern us at all." As one peruses the literature on the subject of inbreeding effects on man in existence in Garrod's time, one can well appreciate the truth of these disparaging remarks; unfortunately, these comments would also apply to a portion of the literature which has accumulated since 1900. Happily, however, the last decade has seen this problem approached in a more critical and objective manner. It is the purpose of this presentation to consider briefly the present status of our knowledge in this area, and to indicate what would appear to be profitable avenues of investigation for the future.

The theoretical basis for anticipating an inbreeding effect appears to have been first advanced, at least in Mendelian terms, by Bateson (1902), and first applied to Garrod's findings (1902) with reference to the incidence of alkaptonuria. Numerous individuals (e.g., Robbins, 1918; Jennings, 1916; Wright, 1921; Fisher, 1949; and Malecot, 1948) have subsequently extended or given added mathematical sophistication to the argument. The most recent development in this area has been the demonstration by Morton, Crow, and Muller (1956) and Kimura (1958) of a means whereby one can estimate the burden of deleterious genes from data on inbred individuals. It is not our purpose to review these theoretical considerations nor to catalogue those rare recessive anomalies in man where it has been possible to demonstrate an increased frequency of consanguineous marriages among the parents of affected individuals as contrasted with the frequency of consanguineous marriages in the general population from which these individuals have been drawn. Rather we are concerned here with the modern studies bearing on the more subtle effects of inbreeding or, if you will, on those characteristics of the individual where the mode of inheritance is unknown or poorly understood.

One can hardly consider the problem of inbreeding effects on man without devoting some attention to the social or cultural factors which give rise to the opportunities for, and the attitudes toward, consanguineous marriages. Since all societies known to us are, to some degree, unique, we may assume that the factors which govern the frequency of

consanguineous marriages in different cultures and at different times will also be, to some degree, unique. That these factors in some instances may and in others definitely will influence the genetic variation in a population can hardly be gainsaid. Unfortunately, we know far too little about these factors to permit us to construct more realistic mathematical models of the inbreeding behavior of most human populations. Permit me to make a stab at illustrating how our lack of knowledge of what might be termed "patterns of mate selection" can lead us astray. In the estimation of "lethal equivalents" (a lethal equivalent is defined as a group of mutant genes of such number that they would cause on the average one death, e.g., one lethal mutant, or two mutants with 50 per cent probability of causing death, etc.), we generally are forced to treat the individuals through whom the relationship in a given consanguineous marriage stems as if these individuals were themselves not inbred. We know that this cannot always be true because the evidence points to the fact that in virtually every country inbreeding was, in the past, far more common than now. If, however, the amount of inbreeding which is undetected is uniformly distributed through the various types of matings, then no bias would be introduced into the estimate. If, however, the undetected inbreeding is a function, as it were, of the detected inbreeding, we could be led to rather erroneous notions regarding the number of lethal equivalents. To support this thesis, we have some evidence that the undetected inbreeding may not be uniformly distributed in Japan. That is to say, we have data which suggest that certain families now and in the past have practiced consanguineous marriages whereas other families have a tradition of avoiding consanguineous marriages. We have no information on the frequency of these two patterns, but the effect of these contrasting patterns on the estimate of lethal equivalents can be easily demonstrated. I do not mean to imply, here, that undetected inbreeding is one of the major inaccuracies in our present estimate of lethal equivalents; this source of bias is advanced solely to illustrate one possible effect of our lack of knowledge of cultural factors on our estimates.

Let us turn now to matters more concrete, namely, the data which are available on pregnancies occurring to related parents. Since time will not permit a comprehensive review of all of the data collected in the various modern studies on inbreeding effects on man, we shall limit our attention largely to the data available on mortality between conception and the attainment of adulthood. The available data (Sutter and Tabah, 1953, 1955; Böök, 1957; Schull, 1958; and Slatis, Reis, and Hoene, 1958) are summarized in Table 1. Before we consider these data, certain definitions and/or explanations of the entries in this table seem in order. Firstly, the term "early fetal death" encompasses miscarriages and spontaneous abortions. A strict distinction between these two events (abortions occurring prior to the fourth month of gestation; miscarriages in the 4th, 5th, or 6th month) has not, in general, been maintained in the various studies. The rather low values observed in Hiroshima with reference to this variable are largely a result of two things. (1) These are terminations occurring prior to the 21st week of gestation, and hence not all miscarriages are included. (2) The data stem from an effort to contact every woman in Hiroshima who aborted a fetus and was seen

TABLE 1

A SUMMARIZATION OF THE OBSERVATIONS ON FETAL AND LATER MORTALITY AND PARENTAL RELATIONSHIP

	Relationship	France						Japan				Sweden		U.S.A.	
		Finistère		Loir-et-Cher		Morbihan		Hiroshima		Nagasaki					
		m	N	m	N	m	N	m	N	m	N	m	N	m	N
"Early fetal death"	Unrelated							1,420	27,139			27	194	25	194
	Second cousin							32	412			---	---	---	---
	1½ cousins							18	329			---	---	---	---
	First cousins							53	972			17	220	36	248
Stillbirths	Unrelated	16	817	14	1,117	34	1,628	345	25,719	443	29,940	8	167	2	169
	Second cousins			5	240	16	309	6	380	9	629	---	---	---	---
	1½ cousins	16	491	3	105	1	78	6	311	3	408	---	---	---	---
	First cousins			8	282	25	461	19	919	22	1,565	5	203	3	212
Death between birth and age of reproduction (20 years)	Unrelated	86	801	82	1,103	176	1,594	31	567	---	---	44	198	4	167
	Second cousins			23	235	39	293	8	144	---	---	---	---	---	---
	1½ cousins	89	475	4	102	19	77	9	106	---	---	---	---	---	---
	First cousins			42	274	90	436	41	352	---	---	20	159	17	209

by an obstetrician in the years 1948-1952. We know that we fell far short of this goal, and, moreover, that not every woman who aborts will be seen by a physcian. There is, however, no evidence that the frequency with which abortions were reported to us was a function of parental relationship. Secondly, the category "death between birth and adulthood" will cover somewhat different intervals of time. Thus, the data from Hiroshima actually span only the first eight years of life; in the data of Slatis *et al.* from the United States, only 10 per cent of the consanguineous children and 20 per cent of the control children had reached or passed their 20th birthday at the time of interview (c. 1957). In the Swedish data of Böök, somewhat more than 60 per cent of the liveborn children of consanguineous parents would have attained their 20th birthday, but only about 20 per cent of the liveborn children of unrelated parents would have reached this age at the time of examination (1947). In the French data of Sutter and Tabah, it is not possible to indicate the proportion of children who attained their 20th birthday. Some of the individuals recorded as dead in Loir-et-Cher and Morbihan are known to have died between the 20th and 30th years of life—one further difference among the observations on mortality presented in Table 1. Finally, the data from Finistère are classified only as consanguineous and unrelated, and not by degree of relationship. In the analysis of the latter data, we shall use the average coefficient of inbreeding among related marriages for marriages occurring in Finistère in the years 1919 through 1929 as a measure of the degree of homozygosity of the inbred children.

In Table 2 we give the intercepts and

TABLE 2

THE EFFECTS OF INBREEDING ON MORTALITY AT VARIOUS STAGES FROM CONCEPTION TO ADULTHOOD

Population	*"Early Death"*		*Stillbirth*		*Later Death*	
	A	B	A	B	A	B
Finistère			.0196	0.389	.1074	2.078
Loir-et-Cher			.0134	0.272	.0725	1.113
Morbihan			.0230	0.529	.1118	1.621
Hiroshima	.0499	0.066	.0134	0.123	.0543	1.007
Nagasaki			.0156	—0.026		
Sweden*	.1392	—0.990	.0479	—0.373	.2222	—1.542
U.S.A.	.1289	0.261	.0118	0.039	.0240	0.917

*See text.
A = intercept; B = regression coefficient.

regression coefficients obtained by fitting a weighted linear regression of the frequency of deaths on the coefficients of inbreeding. The values of A, the intercept, and B, the regression coefficient, given here will differ inappreciably from the intercepts and regression coefficients one would obtain by regressing the logarithm of the proportion of survivors on the coefficient of inbreeding. It need hardly be pointed out that because of the differences in the studies mentioned earlier, these values are of importance only as guides to our thinking, and cannot be viewed as strictly comparable.

As one scans Table 2, the similarity of the values of B within any given mortality category is, with the exception of the Swedish data, striking. In point of fact, the values are more similar, perhaps, than many of us would have anticipated in view of the marked differences in medical practice, nutrition, etc. in the areas for which the data are reported. An alternate, and perhaps more meaningful, way of looking at these values is through the ratio of B/A, that is of the ratio of the inbred to random load (see Crow, 1958). The same similarity persists with this approach. Before we launch into speculations on the significance of this similarity, let us examine the one exceptional body of data, namely, that from Sweden. These data are perplexing, for if they are accepted at face value they suggest a definite advantage to marrying one's cousin. Personally, I'm inclined to doubt the representativeness of the control sample with respect to mortality in families where the parents are unrelated. This is certainly no reflection on the care with which the data have been collected but rather an assertion that sampling vagaries do occur. Since I have made this charge, I feel obliged to indicate at least one of the reasons why I believe this to be so, and why, consequently, one cannot place a great deal of confidence in the estimate of the lethal equivalents which one derives from these data. Among the 32 control marriages, 30 have produced one or more pregnancies. But among the 30 mothers, three would qualify as "habitual aborters" if we accept the common definition, namely, three or more consecutive abortions. Each of these three women has had four or more abortions, and more than 50 per cent of their pregnancies terminated in non-viable offspring. Moreover, these three women account for 48 per cent of the spontaneous abortions and 38 per cent of the stillbirths scored for the control. The bulk of the difference in "fetal deaths" and stillbirths between the consanguineous and control groups can be attributed to these three women. Along

these same general lines, it is interesting to note that a fourth of the 30 mothers has had 12 pregnancies of which 5 terminated in a stillborn infant and one in a spontaneous abortion. While this family is excluded, and perhaps rightly so, from Böök's tabulations because of the occurrence of hemophilia and the possibility that this may be the cause of the numerous stillbirths (4 of which were male infants), we are concerned here with the likelihood of drawing a sample of 30 reproducing women of whom 4 would have the peculiar reproductive performances exhibited by the ones here enumerated. Pregnancy wastage of the order seen in these four women is not common in the United States, and I would imagine not common in Sweden. If, however, we accept this sample at face value, we would be led to conclude that something approximating 10 per cent of women lose 50 per cent or more of their recognizable pregnancies. This does not jibe with our knowledge; the simplest explanation is that the sample is not representative. Böök's data point up the dangers in applying the method of Morton, Crow, and Muller (1956) to data on families of varying sizes where all of the pregnancies for each and every family are scored. Ideally, the method would be applied to data wherein each marriage was represented by one child randomly selected from the sibship. There is, after all, inherent in the regression technique the assumption that observations on the dependent variable are uncorrelated. When families are represented by more than one child and the dependent variable is under some genetic control, this assumption cannot be strictly satisfied because of the correlation in genotype of siblings. In large collections of data, the error introduced by failing to satisfy this assumption will undoubtedly be trivial. In small samples, this may not be so.

As one continues to study the estimates of B, one cannot help but be impressed by how much larger the effects appear to be on the liveborn children as contrasted with those pregnancies which fail to give rise to a viable infant. It is true that if one introduced a time factor into these estimates then a relatively greater number of lethal equivalents would lead to death in the first nine months following conception than would appear to be true for any other nine-month period in life. But if, now, one attempted to relate this notion of the time at which a lethal equivalent operates to the number of deaths (and here we would include spontaneous abortions as deaths) which occur in a given interval, one could be led to the conclusion that among fetal deaths far more are fortuitous than are genetically significant, in the sense of selecting for or against a particular gene or constellation of genes. At least two difficulties in the too ready acceptance of this conclusion present themselves. Firstly, this would assume that the average number of genes which one associates with a lethal equivalent was invariant. This may or may not be so; certainly at the moment we have no evidence one way or the other. Secondly, at a time in life when survival is as intimately associated with the mother as is true of a developing fetus, survival of the fetus may be as much, if not more, a function of maternal genotype than of the fetal genotype. One can readily enumerate reasons why the maternal phenotype could be of very great importance in fetal survival. Thus, it certainly does not require a stretch of one's imagination to believe that successful implantation of a zygote is markedly

influenced by the mother. Furthermore, we know that such critical items as electrolyte balance in the fetus is closely dependent upon maternal electrolyte balance, and that fetal exposure to rubella, toxoplasmosis, etc. are mediated through the mother. The data which we have presented thus far would not, however, be pertinent to this thesis since the importance of the maternal genotype would not necessarily be reflected in data on inbred children. It would, however, seem possible to test the dependence of fetal survival on maternal genotype. Data on the reproductive performance of women who were themselves inbred should provide evidence on the validity of this hypothesis. Such data could be obtained with not much more difficulty than one now obtains data on the outcome of pregnancies to related parents. We have, as a matter of fact, some data bearing on this from Japan. The numbers are small, however, and the only deaths which would be ascertained in the first nine months following conception are those which would occur after the 20th week of gestation, too late to include the bulk of fetal deaths. We present these data in Table 3. The

TABLE 3

THE EFFECT OF AN INBRED PARENT ON THE FREQUENCY OF STILLBIRTHS. A STILLBIRTH IS HERE DEFINED AS A PREGNANCY TERMINATING IN A NONVIABLE OFFSPRING AFTER THE 21ST WEEK OF GESTATION

	Births	Stillbirths	%
Spouses unrelated, wife inbred	130	3	2.31
Spouses unrelated, husband inbred	62	0	–
All unrelated spouses, one or both parents may be inbred	55,659	788	1.42

manner in which these data were collected has been described in detail elsewhere (Neel and Schull, 1956; Schull, 1958). Information as to relationship on the part of the spouses was obtained as a matter of routine on every registered pregnancy in Hiroshima and Nagasaki. Information regarding whether the spouses were themselves inbred was obtained, however, only when a "long form" was used. This latter form was completed on every abnormal pregnancy and routinely on every tenth registration irrespective of whether the pregnancy terminated normally or abnormally. The observations presented in this table are drawn only from the data available on every tenth registration. Clearly one cannot build much of a case on these data; it is interesting, nonetheless, that limited as these data are they do not contradict the hypothesis that fetal survival is dependent to a large extent on maternal rather than fetal genotype.

In the remaining time, let us direct our attention toward the data available on the frequency of infertility in related marriages as contrasted with that in a suitable control. Only three studies provide evidence on this point. In Sweden, Böök found one infertile marriage among the 34 first cousin marriages and two among the 32 control families. Clearly the numbers are too small to be particularly meaningful; however, Böök did attempt to separate voluntary from involuntary sterility. In the infertile marriages which he reports, in two instances there was a clear organic basis for the infertility, namely, testicular atrophy, and, in the third, there was strong presumptive evidence of an organic basis. In the United States, Slatis *et al.* report that in 17 of 109 consanguineous and 11 of 83 control couples the female was not known to have been pregnant. Somewhat more suggestive are their findings when attention is limited to those infertile marriages of

at least ten years' duration. In the latter instance, 15 of 107 consanguineous and 5 of 77 control couples were without known pregnancies. This difference is not significant, however. Sutter and Tabah (1952) find that in Morbihan 5.57 per cent of the control and 8.40 per cent of the consanguineous marriages are infertile, and in Loir-et-Cher 5.96 per cent of the control and 10.61 per cent of the consanguineous marriages are without known pregnancies. The latter difference is significant. Unfortunately, Sutter and Tabah, as Slatis and his coworkers, fail to distinguish between voluntary and involuntary sterility. I, personally, believe that it is quite possible that in Western countries a higher proportion of related couples deliberately refrain from childbearing than would be true for unrelated individuals.

While the evidence is far from conclusive that a higher proportion of consanguineous marriages are involuntarily infertile than is true of unrelated marriages, let us assume that this is so and explore several possible explanations. One possibility is that there is an increased incidence of sterility on the part of one or the other of the related spouses. At first glance, one is inclined to reject this notion on the ground that there is no reason to postulate a higher incidence of, say, anatomical impediments to conception. This is not necessarily so since as we have intimated, individuals who select relatives as their spouses may, on the average, be more inbred than individuals who marry individuals unrelated to themselves. There could, then, be a genetic basis for postulating an increase in parental sterility. Another possible explanation is an incompatibility between parents who are themselves physiologically normal. Specifically, I am thinking of the demonstration of Gershowitz *et al.* (1958) of the presence of hemagglutinins in cervical secretions. We would not, however, expect this to increase infertility on the part of related spouses since these individuals would be antigenically incompatible less frequently than unrelated individuals because of the correlation in genotypes of relatives. The same would appear to be true for histoincompatibility genes. A third possibility is that the apparent increase in infertility stems not from an inability to conceive but rather an inability to support the fetus. Otherwise stated, an increase in the probability of a pregnancy terminating very early, prior to the normal recognition of pregnancy, could account for the apparent infertility. This would be difficult to measure; however, Slatis *et al.* (1958) have suggested one possibility, namely, that the interval between cohabitation and the first recognizable pregnancy may be altered within those families where a recognizable pregnancy occurs. Unfortunately, Slatis' data do not clearly reveal that this occurs nor, for that matter, do the more extensive Japanese data (Schull, 1958) give strong support to this notion. Other more complex hypotheses can be constructed to account for the infertility, but it is questionable whether these would be supported by the limited data at hand. This is surely an area worth exploring, and perhaps from the increasing concern over pregnancy wastage in the United States may come clues to direct our activities.

To summarize these speculations, it seems fair to say that consanguinity studies have already contributed to our understanding of the burden of recessive deleterious genes in a population and may be expected to contribute more as the number of studies increases. One may also say that consanguinity studies

afford opportunities to explore the dependence of fetal survival on maternal genotype and a variety of other genetic hypotheses.

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DISCUSSION

RICHARD LEVINS*

Dr. Schull has shown clearly that there is an inbreeding effect, a generally positive correlation between inbreeding and the frequency of defective development culminating in premature death at one or another stage of growth. The genetic mechanisms underlying this effect are not established, but certain general considerations can guide our speculation in this direction.

Death is not a simple phenotype. In other organisms, we know of many lethal genotypes which cause death in different ways. If a particular gene substitution confers a certain probability of death, and a second dose of the same gene increases the probability by the same amount—that is, if there is no dominance—inbreeding will have no effect on the frequency of deaths. If the lethal is a single recessive, the frequency of death increases linearly with the coefficient of inbreeding. If lethality results from some incompatibility between fetus and mother, inbreeding would reduce the frequency of deaths. Finally, there are lethal genotypes which depend on the interaction of many genes, such as those synthesized by Dobzhansky (1959) by allowing recombination to oc-

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cur between two more or less normal chromosomes. When lethality is the result of the interaction of many genes, the frequency of death may be related to inbreeding in a complicated way.

We know even for clearly defined characters that similarity of phenotype does not imply similarity of genetic mechanism. For something as vague as lethality, this should be even more the case. Thus, although the total lethality may be comparable in different places, the genetics of it may vary. Thus the discrepancies between cities in such studies are not surprising. On the contrary, the similarity looks like phenomenally good luck.

One way of discussing the genetic mechanism is to look for the functional relationship between frequency of deaths and coefficient of inbreeding. With only four values of *F* and the sample sizes available, any attempt to fit a fancier regression than simple linear is unlikely to be successful. When we look at the whole set of data in a different way, a marked nonlinearity is suggested.

Dr. Schull's present paper is based on data published in 1958 from which our rough analysis is taken. Schull (1958) lists 13 comparisons of consanguinity classes by city and type of defect. For each comparison we rank the four consanguinity classes according to decreasing frequency of the harmful effect, and for each consanguinity class we sum the ranks over all comparisons. (Two comparisons, early death and stillbirth, overlap. This does not alter the results) The table below lists for each consanguinity class the expected value of the sum according to two models, and the observed sums. Model A assumes no inbreeding effect, with order completely at random. Model B assumes strict linear relation with no random deviation. In the first case the variance of the sum is 65/4, in the second zero. A realistic model which allows for random deviations from an expected linear order will have expected values between those shown from A and for B, and a variance smaller than that for A. We tested using the variance of A.

Consanguinity Class	Expected A	Expected B	Observed
First cousin	32.5	13	20
1½ cousin	32.5	26	42
2nd cousin	32.5	39	32
Unrelated	32.5	52	36

The deviation of the observation for 1½ cousin, is significant. In 9 out of the 13 comparisons, 1½ cousins showed fewer defects than the 2nd cousins and unrelated categories. If we combine the data into 6 phenotype groups, 1½ cousins show fewer defectives in four of them.

Nonparametric tests of this kind are suggestive of what we might find with more data. No claim is made that nonlinearity has been proven, but only that it is worth looking for.

The reason we are interested in nonlinearity is as a possible clue to second order selection of modifiers that suppress harmful effects of homozygous genes.

The effect of inbreeding is to increase the frequency of all kinds of homozygotes. If deleterious homozygotes are rare in a population, as in large outbreeding organisms, their occurrence will have no appreciable influence on selection. If they are common, as would be the case in inbreeding and self-fertilizing species, they could contribute an appreciable load of harmful effects. We might expect that the population will not take this lying down. Any modifiers which reduce the harmful effects will have some selective advantage and may become important.

We have evidence from *Drosophila* that when a mutant which is moderately harmful, e.g. eyeless, is cultured in the homocygous condition, the eyeless phenotype will tend to be suppressed. When two lines are crossed which have independently suppressed the mutant, the full expression reappears. Dobzhansky found a striking case (unpublished) in which a chromosome which was being propagated in the homozygous condition was actually lethal when homozygous against another genetic background. (See Schmalhausen, 1949 for discussion of this.)

Thus such polygenic suppression is not pure speculation. *Drosophila* and corn are usually outbred and therefore would not normally show the effects of such second order selection. Thus it is not surprising that an approximately linear relation of effect with inbreeding is obtained. However, in man much of the past evolution was in small populations and inbreeding must have been appreciable. Thus we are justified in looking for such effects.

If lethality is caused by a recessive or partly recessive gene, modifiers which ameliorate the harmful effect will reduce the selection against the gene. Since gene frequency depends on the balance between selection and the rate of mutation, gene frequency will increase. Thus the net effect will be to increase the frequency of the bad gene, reduce the effectiveness of the gene, leave the frequency of the phenotype unchanged, and make it more difficult for the geneticists to detect the gene. If lethality is a result of homozygosity for either (or any) allele, and the heterozygote is superior, the selection of modifiers will leave the gene frequencies unchanged but reduce the incidence of the phenotype in the population.

Since polygenic systems such as those postulated are broken down by outcrossing and take very long to build up, one might expect that old isolated populations would show more suppression of inbreeding effects (and nonlinearity) than would races of recent origin.

We are still a long way from having the data or the standardization of collecting techniques that would permit comparisons of populations or satisfactory detection of non-linear inbreeding effects. But we are more likely to find such things if we consider that they may occur than if we too readily attribute queer data to sampling vagaries and statistical artifacts.

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Published 4 times a year, October, December, March, May

For THE UNIVERSITY OF NORTH CAROLINA PRESS

By THE WILLIAMS & WILKINS COMPANY

428 E. Preston St. Baltimore 2, Maryland

Afternoon Session

SPECIAL REPRODUCTIVE PATTERNS IN THE PRESENCE OF PSYCHIATRIC FAMILY PROBLEMS

Chairman: PAUL H. HOCH, M.D.
Commissioner of Mental Hygiene, State of New York
Professor of Clinical Psychiatry, College of Physicians and Surgeons,
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Introductory Remarks by Chairman:

It is a great pleasure and privilege to be invited to participate in the meeting of the American Eugenics Society. The high quality of the papers which will be presented is an assurance that the Society is very much interested in extending our knowledge in this important field.

In eugenics we see a healthy trend to grapple with complex problems on the research level before implementation is asked for or recommended on a practical level. Unfortunately this was not always the case in the past. Quite often in the past before certain hypotheses or assumptions were substantiated they were applied in practice. Soon after they were introduced some of these hypotheses had to be withdrawn or replaced when new knowledge was gained. This, of course, left the practical application of some of these eugenic principles in a confused state. We strongly feel that the practical application of eugenics should be done especially in psychiatry in an unassailable fashion. This means that the practical application of eugenics should be applied only in cases where reliable and confirmed knowledge is at our disposal to make eugenic recommendations

The papers this afternoon deal with the important problem of mating and fertility patterns. The understanding of these patterns will lead to a better formulation of eugenic principles. We are especially interested in the relationship of these patterns to the functional psychoses and to mental retardation. The eugenic approach to schizophrenia and to manic-depressive psychosis will also depend to a very large degree as to how successful we will be in the future in treating these disorders. If treatments can be found which will cure these persons, this would influence the eugenic approach considerably. In the future there will have to be a constant interplay between therapeutic endeavors on one hand and prophylactic endeavors on the other. They will influence each other—at least in my opinion—far more in the future than has been the case until today.

FERTILITY TRENDS IN SOME TYPES OF MENTAL DEFECTS

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Differential fertility is one of the most efficient mechanisms by which a change in the genetical composition of a population can be produced. Such an effect, of course, will materialize only if this differential is strongly correlated with a genetical trait. Furthermore, fertility must imply effective fertility or fitness, i.e., refer to the number of children who survive long enough to marry and produce a new generation. It should be observed that this biological fitness has very little to do with social fitness. In fact, many so-called socially unfit individuals enjoy a high biological fitness. The association between social fitness and biological fitness has turned out to be much more complicated than was anticipated in the past, especially in the older eugenic literature.

In medicine and public health a new field is gaining ground, i.e., the epidemiology of genetical diseases. The development in this area is based on an integration of ordinary methods of epidemiology and population genetics. In this context, the effective fertility of carriers a pathological mutations is one of the most important variables for the prediction of the future epidemiological situation of the population. Other well-established variables are migration, mutation and genetical drift.

There is much need for information on fertility trends for a large number of individuals affected with genetical diseases and for healthy carriers of pathological genes. The data which are available at present are scanty and often unreliable. Furthermore, conclusions based on such data can hardly ever be generalized since fertility trends are greatly influenced by different social, political and public health conditions. One of the objects of this meeting should be to bring out the necessity of collecting and analyzing such data from various populations.

MENTAL DEFICIENCY

Substantial gaps in our knowledge about the causes of mental defects prohibit a more detailed interpretation of known fertility trends. Mental deficiency refers to a certain kind of human behavior which may have any number of causes. Some well-defined clinical entities which presumably are also genetical entities have been separated, such as microcephaly, amaurotic idiocy and phenylketonuria. Others, like mongolism, are relatively well-defined, but the cause is obscure. The large mass, however, must be considered to lack diagnoses, provided we want to maintain some sort of scientific pride in our diagnoses. Eventually this group will be broken down into distinct entities characterized by their main variation components, environmental, major gene and polygenic differences.

The well-defined types of mental retardation are characterized by distinctive neurologic signs, specific metabolic errors, or by both. On the whole, the fertility of such affected individuals is very low or nonexistent. Insofar as these conditions are determined by single gene differences, selection against them is pronounced. If their frequencies remain constant in a population, selection must be counteracted by new mutations or perhaps also by a higher effective fertility (selective advantage) of healthy carriers.

While the implications of the fertility trends of mental defectives without known specific symptomatology must be rather vague, some conclusions nevertheless seem justified. Data collected by Dahlberg (1951) from four counties in Sweden indicated a very low fertility for certified mental defectives, although no control data were presented (cf. Table 1). I have collected more rigorous data

TABLE 1

FERTILITY OF ALL CERTIFIED MENTAL DEFECTIVES OVER 19 YEARS OLD FROM 4 RURAL REGIONS (TOWNS EXCLUDED) IN SWEDEN.

		No. children/individual	
Unmarried	1,258	0.06	0.11
Married	18	3.60	

from three smaller rural communities. Here all individuals with well-defined conditions and with evidence of a specific environmental or genetic origin have been excluded. The remaining group must frankly be called severe mental deficiency of unknown origin. There is evidence in this and other similar materials that a significant genetical component is involved in the variation. To what extent this component is of major gene or polygenic nature cannot be decided at the present time. Matched with comparable control individuals, the mentally deficient had issued 0.7 children per individual and the controls 3.7. Some other details are seen in Table 2. With

TABLE 2

RELATIVE FERTILITY OF A COMPLETE SAMPLE OF MENTAL DEFECTIVES OF UNKNOWN ORIGIN FROM THREE SWEDISH RURAL COMMUNITIES

	No. of individuals	*No. of children*	*No. of children per individual*	
54 Mental defectives				
Extramatrimonial	54	8	0.15	0.7
Intramatrimonial	7	32	4.60	
108 control individuals				
Extramatrimonial	108	16	0.15	3.7
Intramatrimonial	85	384	4.50	

the high standard and effective supervision of public health in Sweden, there is no reason to assume any substantial differences in mortality due to neglect between the two categories. Consequently this group of mental defectives must have had a relatively low effective fertility. In this material, as well as in Dahlberg's, there are a few mentally defective women with many children representing the category which always turned up in the biased examples of the old eugenical misanthropists.

While most people would agree that pronounced mental deficiency could not very well, for mental as well as for physical reasons, be associated with an unduly high fertility, some would still maintain that this is the case for individuals with mild intellectual defects.

In passing, I should like to mention a rather neglected study by Juda (1934) from Germany. She examined the fertility of a group of 97 individuals classified as intellectually inferior on the basis of their school records. They were compared with a group of 85 individuals with normal school records. The net fertility was 2.5 and 2.3 children per

individual, respectively. The children from the inferior group had a significantly higher mortality by the age of 5 years. There would be a lot to say about the detailed interpretation of a study of this sort, but, at any rate, it does not support the idea of a high effective fertility even for individuals with less severe intellectual defects.

Reliable data on fertility trends for individuals with mental defects seem to show that effective fertility decreases with decreasing intellectual capacity. From a statistical and genetical point of view, there is no evidence that this group is replacing itself at the expense of the rest of the population. However, there are many problems which need clarification before any reliable predictions about the future epidemiological trends in genetical mental defect can be made.

HUNTINGTON'S CHOREA

Because Huntington's chorea is a well-recognized entity caused by a dominant gene mutation, fertility trends in this condition can be discussed in more detail. This problem has a particular interest because Reed and Palm (1951) claimed that affected individuals display an increased fertility. Their reported data do not allow a re-evaluation of the conclusions, which, for several reasons, seem doubtful. A comparison was made between the average number of children from affected individuals (6.07 ± 0.9) and from unaffected sibs (3.33 ± 0.5). It is, however, necessary to consider the fact that this disease has a relatively high age of onset. The data, therefore, will be biased in favor of older families with a relatively long marriage duration and a larger number of children. The control data must be adjusted in accordance with this.

I have tested the hypothesis of an increased fertility on data from Sweden collected at this institute by Sjögren (1935). Sjögren does not deal with this particular problem in his report. In 28 families with an affected mother, there were 6.2 children per marriage, and in 26 families with an affected father, 6.1. In total, the 54 families averaged 6.1 children. The marriage rate in this condition does not deviate from the average rate in Sweden. To a great extent, this is probably due to the fact that the average age of onset was 41 years. The data were collected in two Swedish rural communities and represent a complete survey. The fertility data on the choreics have, as far as possible, been matched with data comparable with regard to time, geographical location, social class, duration of marriage and the age of the wife at the marriage. The figures given above refer to marriages in which the wife was 45 years old or over (one exception, where the wife was 42). Disregarding other factors which may influence the number of children, it is justified to compare the figures with a standard that was calculated from the official census of 1930 in Sweden. The result based on 11,944 families with 73,581 children was 6.2 children per family.

At an anthropological investigation carried out at this institute during 1922-24 and comprising about 47,000 conscripts aged 20-22, data were also obtained on the number of siblings. There were 1,372 conscripts from the same county in which Sjögren's survey took place. These individuals belonged to families with an average of 6.7 children. This slightly higher figure is explained by the fact that the duration of the marriages for this group is no less than about 20 years. With the same duration, the census data from the same region gave 6.9 children per family.

Consequently, the Swedish data on Huntington's chorea do not indicate an increased fertility for individuals with this disease when their total marriage reproduction is taken into account. It is likely that their fertility was little affected under the environmental conditions prevailing in this particular population and at the time of the survey. These results agree with those of T. C. Reed (1958) on choreics from Michigan, USA.

THE HEREDITARY ATAXIAS

The Swedish data on these conditions were also collected by Sjögren (1943). They represent what is probably a complete epidemiological survey of the whole country.

Friedreich's ataxia, with a low age of onset (13 $\pm$ 0.7 years) and very likely a recessive type of inheritance, is associated with low fertility. Only 9 children were issued by 83 patients.

Cerebellar ataxia (Pierre-Marie), with a higher age of onset (34 $\pm$ 1.9 years) and probably a dominant type of inheritance, is characterized by a slightly higher fertility than is the case in Friedreich's ataxia. In 53 families, a total of 33 children were born. All of these children belonged to 9 families, giving a fertility rate of 3.7 children in families producing at least one child. Only about 20 per cent of the patients were married. For both of these conditions, it should be safe to conclude that their fertility is below average.

In conclusion, the data from Sweden which I have had the opportunity to analyze do not indicate the existence of a selective advantage as measured by fertility for individuals affected with major intellectual or neurological defects. While some of these conditions may in this respect be relatively neutral, most of them should be at a definite selective disadvantage. Epidemiological predictions are hampered by lack of information on many crucial details.

For future analyses of fertility data for genetical purposes, I would like to point out that the measurements should be based on the number of years during which the individual has been exposed to the risk of having children. As this risk varies with different environmental conditions, the data must be broken down accordingly. A minimum requirement is to secure data for each individual on the age periods (not just number of years) spent as single, married or divorced, the number of children born and the occupation for each of these periods. Such data will in most countries make it possible to utilize the official vital statistics for fairly accurate estimates of differential fertility. Where suitable vital statistics data are not available, it is necessary to secure as controls a group of unrelated individuals matched as closely as possible to the affected group.

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MATING AND FERTILITY PATTERNS IN FAMILIES WITH EARLY TOTAL DEAFNESS*

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PERSONS WHO HAVE BEEN DEAF since birth or early childhood form an important special population group for the evaluation of current mating and fertility patterns. In the present study, deafness has been defined as a "stress-producing hearing loss . . . making effective auditory contact with the world impossible and necessitating special educative efforts" (Kallmann, 1956). Persons so afflicted are forced by their sensory deprivation into a state of semi-isolation, a circumstance that is bound to affect their potentials for mate selection and reproductivity.

Limited in their cultural and social horizons, especially because of lack of speech development, and shut off in varying degree from the hearing world, deaf persons form a society within a society. Out of choice or necessity, they join in groups that cut across many boundaries, including those conventionally drawn for geographic, ethnic, cultural or socio-economic reasons (Kallmann and Rainer, 1958).

A number of special factors are to be considered in studying the patterns of marriage and parenthood among the deaf. For one thing, although a severe defect characterizes the members of the group, it usually does not interfere with their surviving into adulthood, participating in community life, marrying and having children. Deafness is distributed throughout the land, with a prevalence of about 0.1 per cent in the United States and similar rates in other countries (World Health Organization, 1953). Moreover, in about half the cases the condition is congenital, often apparently gene-borne (Sank and Kallmann, 1956). Whether the frequency of this type of deafness is increasing or dwindling is still undetermined.

Other cases of hearing loss may be precipitated by toxic or infectious influences in early life, although they also may be based on a genetic predisposing factor. In any case, while deafness following meningitis and other infections is decreasing, there are now "a greater number of children who are deaf as a result of diseases of congenital origin, such as the sequelae of Rh incompati-

*This report is the thirteenth in a series of publications on the Mental Health Project for the Literate Deaf, which is being conducted by the Department of Medical Genetics of the New York State Psychiatric Institute, Columbia University, aided by a grant from the Office of Vocational Rehabilitation of the U. S. Department of Health, Education, and Welfare. The report was presented as part of an international conference on "Differentiation in Current Mating and Fertility Trends" which was held by the American Eugenics Society at the New York Academy of Medicine on February 14, 1959.

bility, rubella and premature birth" (Fowler, 1958).

Mating and fertility studies of the deaf population may eventually yield sufficient data to throw light on any changing trend in its size, as well as on problems of the etiology, frequency and distribution of the defect. No matter how useful such research may be for present clinical and counseling purposes, its most valuable contribution would unquestionably be to help lessen the number of persons born deaf in the future.

When it comes to counseling deaf persons and their families, suggestions are usually directed toward securing optimum conditions for successful adaptation in all phases of life. Advice is sought on such matters as whether to marry and when, whether to choose a deaf or a hearing mate from a deaf or hearing family, how many children to plan on, or, having had a deaf child, whether to risk another, and the problem of divorce. The consequences of these decisions combined with other influencing factors, such as type of education, method of communication, and kind of sex instruction, will not only leave their mark on the next generation but will affect the health and happiness of the present one.

These factors are part and parcel of any broad mental health program. In considering any effective population policy it is necessary to take into account the welfare of the parents as well as that of prospective children, always with a view to preserving the family as the basic unit of society (Rainer, 1958b). These goals must certainly be envisioned for such special groups as the deaf, if there is to be any hope of their general attainability.

Curiously enough, in a century of considerable progress in the education of the deaf, there have been very few adequate studies on their mating and fertility patterns. A study by Fay in 1898 provided extensive data on 3078 marriages (Fay, 1898), and census figures for 1900, 1910 and 1920 are cited by Best (Best, 1943; Dept. of Commerce 1928). Despite the need for current data that would reflect a modern psychiatric and genetic orientation, a search revealed no such information on the demographic and health characteristics of the deaf population.

In view of this lack, our pilot study was organized in New York State in 1955 (Kallmann, 1956). The data to be presented here grew out of this project, which encompasses the following general aims:

1. To investigate the adjustive norms and specific family problems of the literate deaf in two statistically representative deaf populations, the total deaf population of the State of New York, and all deaf twins in the eastern half of the United States.
2. To develop and apply improved methods for the psychiatric management of the specific problems of deaf adolescents and adults.

Research subjects for this study were ascertained with the aid of all available agencies, including schools, hospitals, statistical bureaus, and various cultural, social and vocational organizations. Without exception, these groups cooperated in our task of listing the name of every deaf person over the age of 10 in New York State. After almost two years' work, conducted in cities, towns and villages throughout the state, an index of over 12,000 names and addresses was built up.

Within this total frame, various samples were studied by different methods.

A random sample of 300 adults was interviewed at length to determine such qualitative data as sexual maturation patterns and attitudes regarding marriage and parenthood, problems of marital adjustment and maladjustment, the planning of family size and the nature of intra-family relationships. Also, life performance was evaluated in the community, at work, in clubs and associations.

Psychiatric patients, hospitalized or otherwise, were investigated clinically as to diagnosis, course and treatment (Altshuler and Rainer, 1958), and demographically as to the added effect of mental illness on family patterns of the deaf.

Because of the particular value of marriage and fertility data in the project, it was decided to secure this information for the entire group of deaf adults. As the first step in sampling this heterogeneous and often elusive population, a questionnaire was mailed out regarding date of birth and the presence or absence of twins or other deaf persons in the family. Over 20 per cent or about 1700 persons replied, far more than anticipated, very likely because of the innocuous nature of the "Are you a twin?" approach.

All those who answered and a 1:5 random sample of those who did not received a second questionnaire, two pages long, requesting more detailed information on marriage and parenthood. The data to be presented here are taken from the replies to this second questionnaire (about 1000) and constitute a first approximation to a properly weighted random sample of the entire frame (Deming, 1956). Those who did not reply will be followed up by a personal call or through cooperating schools, clubs, and leaders of the deaf.

Comparable data have been obtained on deaf patients in state mental hospitals to determine the combined effect of deafness and institutionalized mental illness, particularly schizophrenia, upon marriage and fertility. By contrast, a small but select number of deaf persons who have achieved professional or executive status furnishes significant information about this special group.

Since the tabulated data are supposed mainly to demonstrate the statistical procedures applied and to point out trends and hypotheses, tests of statistical significance have frequently been omitted. More detailed analyses, particularly from a genetic standpoint, have been postponed until such time as complete data will be available.

Figure 1 represents graphically by age and marital status the first group of 968 deaf persons to cooperate in the study. This diagram reveals that the base of this pyramid is too narrow, most likely because of a deficit of replies from unmarried males and females in the two lowest age groups.

Tabulation of these cases according to present marital status (Table 1) yields a total marriage rate of 61 per cent for males, and of 61.5 per cent for females. The given figures are only slightly lower than those for the total New York State population (66.8 per cent for males, 62.9 per cent for females), but they reflect the previously discussed underrepresentation in the two lowest age groups. The disparity increases if only persons over 24 years of age are compared (66.7 and 78.8 per cent for married males, and 66.8 and 70.4 per cent for married females).

Another interesting trend seems to be indicated by the finding that the current marriage rate of deaf persons (over 60

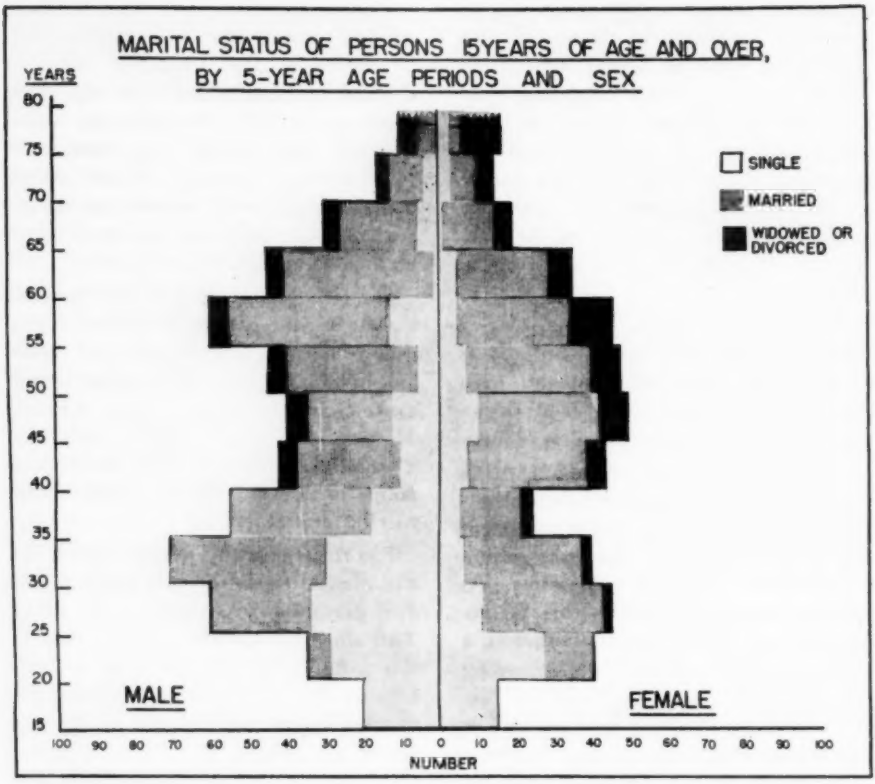

FIGURE 1

per cent in both sexes) is considerably higher than the one reported in 1920 for the total deaf population of the United States (36.1 per cent for males, 39.4 per cent for females). It is in accordance with expectation that in our present sample, the highest celibacy rates are found among hospitalized schizophrenics (82.3 and 61.4 per cent, respectively). While only 10.1 per cent of the males and 24.6 per cent of the females are married, an additional 5.1 per cent of the males and 10.5 per cent of the females are divorced.

Compared with the hearing population, the present deaf sample also shows an apparently increased proportion of divorced persons. This finding may mean that either more deaf than hearing persons are divorcing, or that those who divorce find it harder to remarry. The latter explanation seems more plausible because of the relatively low figure for married deaf persons ever divorced, 5.9 and 9.1 per 100 married males and females, respectively.

A telling factor in determining the choices and family patterns of a deaf individual is the hearing status of his parents. Table 2 separates the responding deaf persons, male and female, into those with neither parent deaf, and those with

TABLE I.
PERCENT DISTRIBUTION OF DEAF ACCORDING TO MARITAL CONDITION

		NUMBER OF DEAF	PERCENT UNMARRIED		PERCENT MARRIED		PERCENT WIDOWED		PERCENT DIVORCED	
			DEAF	GEN. POP.	DEAF	GEN. POP.	DEAF	GEN. POP.	DEAF	GEN. POP.
NEW YORK 1958	MALES, TOTAL	534	33.7	27.6	61.0	66.8	3.2	4.6	2.1	1.0
	MALES 25 AND OVER	478	27.4	14.4	66.7	78.8	3.6	5.6	2.3	1.2
	FEMALES, TOTAL	434	24.2	23.2	61.5	62.9	9.9	12.4	4.4	1.6
	FEMALES, 25 AND OVER	376	16.7	12.5	66.8	70.4	11.4	15.2	5.1	1.8
USA 1920	MALES, TOTAL	—	60.4	35.2	36.1	59.4	2.6	4.8	1.0	0.6
	FEMALES, TOTAL	—	54.4	27.4	39.4	60.7	5.2	11.2	1.0	0.8
N.Y. STATE HOSPITALS	SCHIZOPHRENICS, MALE	79	82.3	61.9	10.1	26.8	2.5	1.2	5.1	9.7
	SCHIZOPHRENICS, FEMALE	57	61.4	34.8	24.6	44.8	3.5	5.6	10.5	14.3

GENERAL POPULATION FROM 1950 CENSUS

TABLE 2.
MARITAL STATUS OF DEAF ACCORDING TO DEAFNESS OF THEIR PARENTS

	MALES			FEMALES		
	NEITHER PARENT DEAF	ONE PARENT DEAF	BOTH PARENTS DEAF	NEITHER PARENT DEAF	ONE PARENT DEAF	BOTH PARENTS DEAF
NUMBER	505 (94.6%)	11 (2.1%)	18 (3.3%)	406 (93.5%)	9 (2.1%)	19 (4.4%)
PERCENT EVER MARRIED	65.9	54.5	83.3	74.4	100.0	94.7

one or both parents deaf. It will be noted that about 94 per cent of our sample are the offspring of two hearing parents, although some of them have deaf cases among siblings, children or other relatives. Obviously, while the deaf do not replace themselves, they do contribute to some extent to the recurrence of deafness in the next generation. A similar conclusion is to be drawn from data on the children of the deaf.

Of particular interest is the high marriage rate of those deaf who have deaf parents. With both parents deaf, the marriage rates are 83.3 per cent and 94.7 per cent for the two sexes; with neither parent deaf, 65.9 per cent and 74.4 per cent, respectively. If this dif-

TABLE 3.
DISTRIBUTION OF SIBLINGS OF THE DEAF BY DEAFNESS OF THEIR PARENTS

	NEITHER PARENT DEAF (910)	ONE OR BOTH PARENTS DEAF (57)
TOTAL NUMBER OF SIBLINGS	2649	124
TOTAL NUMBER OF DEAF SIBLINGS	160	24
PERCENT DEAF SIBLINGS	6.0	19.4

TABLE 4.
PERCENTAGE DISTRIBUTION OF DEAF BY SIZE OF SIBSHIP

	NEITHER PARENT DEAF (911)		ONE OR BOTH PARENTS DEAF (57)	
FAMILY SIZE	0 DEAF SIBS (791)	1 OR MORE (120)	0 DEAF SIBS (39)	1 OR MORE (18)
1	15.5	-	20.5	-
2	21.0	12.5	25.6	27.8
3	18.5	11.7	20.5	33.3
4	14.8	15.0	10.3	22.2
5	10.1	14.2	10.3	11.1
6	8.5	15.8	7.7	-
7	4.8	10.0	5.1	0.6
8	2.7	13.3	-	-
9	2.7	4.2	-	-
10	0.5	1.7	-	-
11	0.6	0.8	-	-
12 OR MORE	0.4	0.8	-	-

ference continues to prove significant, the trend may have negative selective value. It is not clear, however, how such an effect is mediated, through the advice or example of the parents, or through other circumstances in the parental home.

That a detailed genetic analysis will be needed at a later date is shown by the data summarized in Tables 3 and 4. At present, if all cases of deafness are used to compute the percentages of deaf siblings with and without a deaf parent from the total number of siblings available in the survey, there is a difference from 6 per cent to 19.4 per cent according to whether the given sibships had no or at least one deaf parent (Table 3). The application of various corrective procedures in the final analysis (omission of siblings of isolated, adventitiously deaf subjects, expectancy rates by means of the Weinberg method, etc.) will probably confirm that the majority of genetically determined forms of early total deafness follow a recessive mode of inheritance. In any case, the data tabulated so far give some indication of the expectancy of deafness among the children of matings that have already produced one deaf child. Included in the present series are 96 sibships with 2 deaf subjects, 31 with 3, 2 with 4, and 5 with 5.

A tendency toward familial clustering is also revealed by an analysis according to family size (Table 4). Apparently, parents, particularly hearing parents, who have two deaf children, go on to have larger families than those with just one deaf child, perhaps in an attempt to compensate. If this hypothesis is correct, the trend may be more pronounced than the table indicates, since the order of birth has not been taken into account.

In another set of tables, marriage rates for deaf females are combined with fertility rates expressed as children born per 100 mothers. Table 5 divides the group of 434 deaf women into five-year age groups, including 225 women over 45 whose fertility period is considered completed. Because of the previously noted underrepresentation of unmarried persons in this group, the observed proportion of females who have ever been married (75.8 per cent) is probably an overestimate. It is unlikely, however, that the final figure will be as low as the one that prevailed in the earlier years

TABLE 5.

CRUDE MARRIAGE AND FERTILITY RATES OF DEAF WOMEN BY AGE

	AGE	NUMBER OF WOMEN	PERCENT EVER MARRIED		PERCENT OF MARRIED WHO ARE MOTHERS		CHILDREN EVER BORN PER 100 MOTHERS	
			DEAF	GEN. POP.	DEAF	GEN. POP.	DEAF	GEN. POP.
TOTAL DEAF WOMEN 1958	15 and OVER	434	75.8	76.9	74.4	77.1	211	235
	15-24	58	27.6	31.1	62.5	56.0	120	142
	25-34	86	77.9	82.6	71.6	79.0	196	197
	35-44	65	80.0	88.4	78.8	80.3	229	241
	45 and OVER	225	86.2	89.4	75.3	79.0	216	280
1900	15 and OVER	—	30.7	68.8	85.9	92.9	—	—
1910	15 and OVER	—	41.4	70.3	79.0	—	—	—
1920	15 and OVER	—	45.6	72.6	78.0	—	260	330

TABLE 6.

CRUDE MARRIAGE AND FERTILITY RATES OF DEAF WOMEN BY RELIGION, AREA, MENTAL CONDITION

		NUMBER	PERCENT MARRIED	PERCENT OF MARRIED WHO ARE MOTHERS	CHILDREN EVER BORN PER 100 MOTHERS
TOTAL DEAF FEMALE	ALL WOMEN	434	75.8	74.5	211
	ROMAN CATHOLIC	139	66.2	71.7	230
	JEWISH	135	87.4	81.4	190
	PROTESTANT	155	74.2	69.6	221
	NEW YORK CITY	242	73.6	76.4	194
	OTHER URBAN	160	82.5	72.0	227
	RURAL	32	59.4	73.7	257
HOSPITAL	SCHIZOPHRENICS IN HOSPITAL	57	38.6	81.8	211

of the century, when it varied from 30.7 per cent in 1900 to 45.6 per cent in 1920. The fertility rate for mothers also remains below the level of the general population, but relatively less so than in 1920. The current values are 211 children born per 100 deaf mothers, as compared to 235 among the hearing in the 1950 census.

In Table 6, the rates for the total group of deaf women are broken down into differential rates for the three major religious affiliations and for residence areas as defined by the census. At this stage, the usefulness of these data lies mainly in further describing the composition of our responding group. Added to this table are the marriage and fertility figures for all schizophrenic deaf females in state hospitals. Once again, there is a low marriage rate (38.6 per cent), but a fertility rate of 211 per hundred mothers, comparable to the findings for deaf women generally.

Of crucial importance in this study is the particular mating and fertility pattern determined by the presence or type of deafness in both marital partners. Table 7 combines the given data as related to assortative mating and ensuing productivity, and includes an estimate of the differential productivity of deaf children. Interestingly, deaf women married at a median age of 23 to 24, somewhat later than the general population for which the median age is about 21. Only 4 per cent of the women who were born deaf and 13 per cent of those who became deaf early in life were married to hearing men. The rest had husbands who were deaf since birth or early childhood.

In each mating group, fertility rates for mothers are lower than they were in 1898, reflecting the fertility decline in the general population. It should be remembered, however, that the marriage rate of the deaf has risen considerably, with consequent increase in the net fertility for all deaf. Incidentally, hear-

TABLE 7.
CRUDE MARRIAGE AND FERTILITY RATES OF DEAF WOMEN ACCORDING TO TYPE OF DEAFNESS

	PERCENT EVER MARRIED	MEDIAN AGE AT FIRST MARRIAGE	HUSBANDS	PERCENT MOTHERS		CHILDREN PER 100 MOTHERS		PERCENT WITH ONE OR MORE DEAF CHILDREN	
				1958	PAY-1898	1958	PAY-1898	1958	PAY-1898
BORN DEAF (168 WOMEN)	66.7	24	BORN DEAF (25%)	64.3	83.6	178	278	27.8[a]	24.8
			BECAME DEAF (71%)	74.7	84.4	222	265	11.9	8.1
			HEARING (4%)	80.0	89.0	125	311	0	14.7
BECAME DEAF (266 WOMEN)	81.6	23	BORN DEAF (19%)	78.0	84.4	219	265	15.6[b]	8.1
			BECAME DEAF (68%)	72.1	85.9	206	237	3.8[b]	3.6
			HEARING (13%)	82.8	86.8	242	265	4.2[b]	3.2

a- 16.7% HAVE TWO OR MORE DEAF CHILDREN
b- TWO OR MORE DEAF CHILDREN

ing siblings of our deaf subjects have not been used as "normal" controls for fertility, since there is reason for assuming that they themselves represent a selected group, both genetically and socially.

The last columns in Table 7 reveal that over 27 per cent of the marriages where both partners were born deaf, and from 12 per cent to 15 per cent of those where one partner was born deaf resulted in deaf children (in most cases, more than one). A significantly increased number of deaf children was also produced by persons who presumably became deaf during childhood. This group calls for further genetic investigation. In any event, the present data would seem to be sufficient to demonstrate the operation of assortative mating patterns in the deaf, especially the proportionate contribution to the deaf population by parents themselves born deaf.

The extent of this contribution, as shown in Table 8, is about 10 per cent of all children born to our subjects.

TABLE 8.
DISTRIBUTION OF CHILDREN OF DEAF

	TOTAL DEAF – 1958 (502 MARRIAGES)		FAY – 1898 (3016 MARRIAGES)	
	NUMBER	PERCENT	NUMBER	PERCENT
DEAF CHILDREN	78	10.2	588	8.7
HEARING CHILDREN	684	89.8	5091	75.0
UNKNOWN	—	—	1103	16.3
TOTAL	762	100.0	6782	100.0

These fertility figures, when compared with those of Fay in 1898, indicate a steady rate of production of new deaf children. In other words, there is an increased reproduction of deaf children because of the much higher marriage rate. Just as with our findings in the deaf subjects' parents, the number of deaf children may not be enough to replace the deaf population, but it will somehow contribute to an increase attributable to the assortative mating pattern in the deaf isolate.

While the family size pattern of deaf subjects for those with no deaf children and with one or more further illustrates the clustering of deaf in families (Table 9), the fertility of deaf mothers by children per year of fertile marriage is presented in Table 10. The given rate

TABLE 9.
PERCENTAGE DISTRIBUTION OF DEAF BY TOTAL NUMBER OF CHILDREN

NUMBER OF CHILDREN	TOTAL DEAF (883)	WITH NO DEAF CHILDREN (621)	WITH ONE OR MORE DEAF CHILDREN (62)
0	26.2	28.8	—
1	23.1	23.2	22.6
2	32.5	31.4	43.4
3	12.0	11.1	21.0
4	3.4	3.4	3.2
5	1.3	1.1	3.2
6	0.3	0.3	—
7	0.4	0.3	1.6
8	0.3	0.3	—

TABLE 10.
CHILDREN OF DEAF MOTHERS PER YEAR OF FERTILE MARRIAGE

	NUMBER OF MOTHERS	NUMBER OF CHILDREN	CHILDREN PER YEAR OF FERTILE MARRIAGE
ALL DEAF MOTHERS	243	514	.13
MARRIED BEFORE AGE 30	213	468	.13
MARRIED 30 OR OVER	30	46	.14
SCHIZOPHRENIC MOTHERS – TOTAL YEARS	18	38	.14
SAME – OMITTING YEARS IN HOSPITAL	18	38	.23

(.14) is similar to that for the small but complete group of hospitalized schizophrenic deaf mothers whose rate is increased to .23 when their hospital years are excluded. This increase reflects the fact that most of the children were born in the first half of their mothers' fertility period, before the date of first admission at an average age of 32 years.

TABLE II.

MARRIAGE AND FERTILITY PATTERNS OF DEAF "ACHIEVERS"

	TOTAL NUMBER	NUMBER SINGLE	NUMBER MARRIED	SPOUSE	NUMBER OF MATINGS	NUMBER OF CHILDREN PRODUCED[b]
BORN DEAF[c]	9	3	6	BORN DEAF	1	3
				BECAME DEAF	4	0
				HEARING	1	1
BECAME DEAF[a,c]	13	4	9	BORN DEAF	0	0
				BECAME DEAF	2	3
				HEARING	7	14

a. average age 8
median 7

b. all children hearing

c. all parents hearing

Finally, Table 11 summarizes the first results of a study of deaf persons who have achieved status educationally and vocationally that would be deemed outstanding in any population. Among the 22 cases investigated are dentists, chemists, engineers and research scientists, accountants, lawyers and business executives, artists and illustrators. All of them had hearing parents, and all the children are hearing. Except for those who were born deaf, age of onset of deafness was relatively high. With the median age at 7, at least half became deaf after they began to learn language and speech patterns. Of the 15 who married, almost half married hearing mates. No statement can as yet be made about general family patterns correlated with attainment in this special group.

Other subgroups which are still incomplete or otherwise unsuited for comparison with respect to marriage and fertility rates include 250 mentally defective deaf in state institutions, mostly unmarried; some deaf inmates in prisons; and a much larger number passing through the lower courts, not infrequently suspected of homosexual acts.

It is expected that there will be some modification of the data presented here, when the less cooperative families are heard from. Needless to say, the difficulties in communicating with the deaf and obtaining their consent for intensive family investigation have complicated our work. Obviously, however, comprehensive studies of this kind form the basis for a realistic approach to family counseling, and to all psychiatric programs, not only for deaf people but for all groups who are handicapped to a similar extent.

Acknowledgments

In collecting and tabulating the raw data for this report, valuable contributions were made by the following members of our research staff: Mrs. Christel Wolff; Drs. K. Altshuler, G. Baroff, and M. Klaber; and Mr. M. Wodin. The unfailing assistance of our statistical consultant, Professor W. Edwards Deming, is also gratefully acknowledged.

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DISCUSSION

W. EDWARDS DEMING*

The contribution of the statistician lies in the statistical design of surveys and in the interpretation of data derived from statistical surveys. The statistical design of a survey is characterized by the description of a sampling unit (person, household, segment of area), the method of selection, the procedure by which to form the estimates, and the procedure by which to calculate an estimate of the margin of sampling variation for each estimate. The evaluation of reliability requires also detection and evaluation of operational blemishes in procedure, as well as consideration of the margin of sampling variation.

The aim in statistical design is to obtain the maximum amount of reliability that is possible within the limitations of allowable costs, interviewers' skills, and any other administrative restrictions. The aim in interpretation is to extract whatever objective information lies in the data for whatever purposes experts in the subject-matter may deem important; and to evaluate the reliability of the statistical information so extracted.

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It is useful to think of three types of uncertainties in data.

TYPE I. *Uncertainties that are inherent in the questionnaire, or in the method of interviewing, or in the method of test.* These are built-in deficiencies which one might describe briefly as missing the point, or getting information that is not needed for the purpose. They arise from imperfect knowledge of the subject-matter. They do not arise from flaws in carrying out the specified survey-procedure. A recanvass, for example, of areas, people, or households that have been selected for the sample will detect operational blemishes (Type II), but it will not detect built-in deficiencies. Another feature of Type I is that it is independent of the size of the sample. We do not reduce this type of uncertainty by increasing the size of the sample to a complete coverage.

TYPE II. *Blemishes and blunders in carrying out the procedure specified.* Going to testing, coding, computations, and other work. Nonresponse is an important example. These errors have their origin in imperfect workmanship, or in bad luck. One may detect them by a recanvass or by other con-

trols. All of them occur as well in a complete census as in a sample.

TYPE III. *Errors of sampling.* In probability sampling, the margin of uncertainty that sampling introduces is measurable and is never in doubt. In fact, probability sampling is a procedure laid out in advance so that the margin of sampling variation can be calculated from the results of the sample itself. The margin of sampling variation properly calculated is not a matter of opinion, but it is a mathematical consequence, with a standard interpretation. This is the one type of uncertainty concerning which we have, in probability sampling, complete information.

The margin of sampling variation (usually measured by the so-called standard error) unfortunately does not measure the effect of nonresponse nor of persistent omissions, exclusions, or departures from procedure (uncertainties of Type II), and of course it gives no hint of uncertainties of Type I. It does include, however, the variable effects of the interviewers, their variable judgment, and the differences between interviewers as well.

One of the most troublesome uncertainties of Type II is nonresponse. Nonresponses create a problem because the people that respond often have different characteristics from those that do not. Differences may be especially prominent in educational level, income, marital status, occupation, amount spent on travel, and in other important subjects of investigation. The same may well be true of the fertility of the deaf. The only way to discover what the differences are between responses and nonresponses is to investigate a sample of the nonresponses. This is the plan here.

Missing, illegible, or unacceptable information creates about the same problem as nonresponse. Fortunately, missing, illegible, and unacceptable information are often but by no means always fairly well distributed amongst all classes, and when this is so, they create little or no bias.

The responses treated by Dr. Rainer came from 1000 people that had cooperated by replying to a mailed questionnaire for a previous study that took place in early 1958. The following steps are being taken to investigate the nonresponses and to complete the survey:

1. Questionnaires have already been sent to a 20 per cent sample of the people that did not respond a year ago, and whose envelopes did not come back from the Post Office unknown (hereafter, the U.P.O. group). The returns are astonishingly good. A second mailing will doubtless elicit further response. Those that we do not hear from will go back to the original source of references with a request for help.

2. A 10 per cent sample of the questionnaires that came back U.P.O. a year ago will likewise go to the source of reference with a request for help.

3. Finally, there will be a special investigation to try to learn something about the people that the source of reference has lost track of.

A special feature of the samples that are being carried out by the Psychiatric Institute is a replicated design that permits easy calculation of the margin of sampling error. In a replicated design, the sample is not one big sample, but is 2 or more independent interpenetrating subsamples, each one a valid sample of the whole. Replicated designs started with Mahalanobis in India in 1936. Here, there are 10 subsamples. One-tenth the range between the highest and the lowest of the 10 independent results gives a ready estimate of the standard error of the mean of the whole sample, and hence an estimate of the margin of sampling variation. As a matter of fact, one may see without any mathematics at all that the probability is only $(1/2)^{10}$ or 1/1024 that the minimum shown by the 10 subsamples is above the median that would come from the equal complete coverage (a 100 per cent sample carried out with the same definitions and care as were used in the sample). Similarly, the probability is only $(1/2)^{10}$ that the maximum shown by the 10 subsamples is below the result that would come from the equal complete coverage.

Easy calculation of the standard error will facilitate tests of significance between the various waves of responses. Thus, it will be possible to see whether (e.g.) the number of children ever born, or the proportion of women ever married, varies significantly between the 1000 willing responses that Dr. Rainer's paper deals with, and the wave of responses that is coming in from the questionnaires that we sent to a 20 per cent sample of the people that did not respond in the study that was carried out in early 1958, or the information that we hope to get from the sources of referral and otherwise. Comparative studies like these may throw light on the characteristics of the people that respond readily, and those that do not, and the reasons why. Ready ways of computing standard errors may thus contribute not only to our knowledge about the sampling variation but may also aid studies of the biases that arise from nonresponse.

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FAMILIES WITH MANIC-DEPRESSIVE PSYCHOSIS

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WHETHER THERE IS A RELATION BETWEEN affective psychosis on the one hand and marriage and propagation on the other hand has long been a matter of interest to psychiatrists. Pinel, for example, tells us that he was at pains to collect, between 1801 and 1805, statistics on this matter (which he reproduced in a table) but that, not knowing the proportion of single to married at given ages in the general population, he could not draw conclusions from the ratios he found among mental hospital patients: "On ne peut tirer dans les hospices aucune induction du rapport numérique observé entre ces deux états (mariage et célibat), en faveur d'une disposition plus ou moins grande a contracter la manie ou la mélancolie." Nor, he said, could he properly draw any inference from the observation that among women patients with chronic and dementing processes there were many more spinsters than married women: but he suggests that marriage may have a prophylactic effect against inveterate forms of mental illness. Here we have the burden of many a subsequent discussion of marriage in this context. The point was succinctly expressed, eighty years later, by Kraepelin, then at the beginning of his career. "It is undeniable that in very many cases celibacy is to be regarded as the consequence of psychological immaturity or of a past or still active mental illness. But marriage itself may have a prophylactic value, in spite of the risks it entails and the cares it brings with it: it gives greater satisfaction and security and saves people from excesses. It is the widowed and the separated who are most endangered, for they have often to bear nearly all the risks and troubles of marriage without enjoying any of its benefits."

A great deal of what has been written since consists of variations on that theme. It is true that our focus has altered and our field of interest widened: we have concerned ourselves as much with the patient's progeny and contributions to the genetic stock as with his personal well-being and hazards; we have tried to plumb his least accessible motives and dissect his social relations; and we have remembered that not all the mentally ill reach a mental hospital (Lewis, 1958). But the broad question is still being argued, chiefly in connection with schizophrenia. The mating habits and fecundity of those with affective disorders have received far less attention, which is surprising when we consider how manic excitement—or for that matter a sanguine hypomanic temperament—may predispose to heightened sexual activity and unrestraint, and how depression in contrast can militate against libidinal gratification.

Those who have addressed themselves to the problem are not yet unanimous as to whether manic-depressives differ appreciably from the normal population in the extent to which they marry and have children.

Where there is any dispute on this matter, we must all trundle our little wheelbarrows along to the great quarry which Essen-Möller has provided for us. No one has amassed such full and rigorously analyzed material as he in his unrivalled study of the fertility of manic-depressive, schizophrenic and epileptic patients in mental hospitals.

Now Essen-Möller's conclusions about manic-depressives were, summarily stated:

1. Their marriage rate is about normal before their illness; afterwards it falls to about half normal.
2. The length of the period following the illness does not affect the post-illness marriage rate.
3. The mortality rate after the illness is one and a half times higher than before the illness.
4. Before the illness the number of children of the married patients corresponds to that of the normal population: and in his series it diminished *pari passu* with the general birth rate from the beginning of the century onwards.
5. After the illness the fecundity of the married patients is not less than it was before the illness.
6. Extramarital fecundity of manic-depressive women remains the same after the illness as it was before it.

And in general, he concludes

7. "The manic-depressives reproduce themselves about as much or as little as the rest of the population. Their lowered marriage-rate, and their raised mortality rate after the illness, have only a slight effect on their total reproduction because the age of onset for the illness is relatively advanced." (Essen-Möller, 1935)

A more recent investigator, Stenstedt, examined a smaller number of manic-depressives admitted to certain mental hospitals in South Sweden (Stenstedt, 1952). Like Landis and Page (1938), he found that married people were rather less frequent among them than in the general population, due allowance being made for age-distribution; but the difference was not statistically significant except in the married males. As to the fertility of those who married—the proportion of childless marriages and the number of live births for the married women among his manic-depressives were like those in the general population.

In a larger series drawn from mental hospitals serving populous areas of London, my colleague, the late Dr. Vera Norris (1956), calculated the age-standardized ratios of first-admission rates of single to those of married persons with manic-depressive psychosis: for men the ratio was 1.8, for women 1.6. In Ødegaard's data (Ødegaard, 1946) the ratio was 1.5 for men, 1.3 for women. The preponderance of single persons in the London patients was evident in all age-groups except men and women under 30, of whom approximately the same percentage was single as in the corresponding general population of London. The discrepancy was greatest in men between 30 and 49: 34 per cent of the manic-depressive men of this age were single, as against only 17 per cent in the normal

male population of London. For manic-depressive women of this age-group the discrepancy was evident but less wide: There were 472 of them, 29.7 per cent of whom were single, as against 19.9 per cent single in the normal population.

It is fair to conclude from these and similar reported studies that the proportion of single men and women among manic-depressive patients admitted to mental hospitals tends to be higher than in the general population, but that the discrepancy is not gross, and is indeed hardly noticeable in the younger patients who have not yet entered on their fourth decade. Manic-depressive psychosis is therefore strikingly different from schizophrenia in these respects.

This finding in studies coming from several different countries is all the more impressive when we consider the wide variation in the apparent prevalence of manic-depressive psychosis in different regions.

Thus in Canada (Dominion Bureau of Statistics, 1954) manic-depressive patients account for only one-twelfth of all first admissions to mental hospitals, in contrast to schizophrenics who make up a quarter of all first admissions; similarly Dr. Kallmann expressed the opinion a few years ago, that "ordinarily this type of psychosis (manic-depressive) is known to be relatively infrequent, and is certainly less frequent than schizophrenia, even if the former diagnosis is applied quite liberally." (Kallmann, 1952) Yet in England and Wales (Registrar General 1955) it is the manic-depressives who amount to a quarter of all first admissions to mental hospitals, whereas schizophrenics contribute only a sixth of the first admissions. Whether we attribute this divergence to differences in diagnostic practice or to the administrative and social factors that determine admission to hospital, it is remarkable that in spite of it English, Scandinavian and American data about marital status have so far revealed (with one exception) much the same state of affairs, so far as proportion of single to married patients is concerned, or ratio of first-admission rates for single to those for married manic-depressives. It may well be, of course, that data drawn from first admissions do not give a true picture, for Essen-Möller observed that those patients who were judged to be manic-depressive in their first attack, but later found to be schizophrenic, had shown before the outbreak of their illness the same proportion of single to married as manic-depressives generally did, but after the illness behaved in this and other reproductive respects like typical schizophrenics.

The outstanding exception to the general findings about the fertility of manic-depressives is in Dr. Kallmann's twin data. From these he concluded that "manic-depressive twins and siblings marry less frequently, tend more often to remain childless when married, and produce fewer children if fertile, than is true for the non-psychotic persons among their co-twins and siblings, as well as for the general population. In fact, the fertility rates of our manic-depressive index cases are even lower than the corresponding rates of schizophrenic index cases." (Kallmann 1950, 1954) This is rather hard to reconcile with the conclusions from other studies, carried out with corresponding rigor. Three possibilities present themselves: (1) to take manic-depressive twins as propositi biases the sample of manic-depressives, since twins may have different reproductive behavior from non-twins; (2) the sample,

consisting of 85 manic-depressive twin index cases, is perhaps unduly small for this purpose; or (3) the diagnosis of manic-depressive psychosis may have been made on different grounds from those adopted by Essen-Möller and other investigators. The first and third of these possibilities are evidently frafetched and unacceptable. We have no reason to suppose that twins, qua twins, marry less than other people or have fewer children, and Kallmann was exceptionally careful in applying, and explicit in stating, his criteria, both positive and negative, for diagnosing manic-depressive psychosis. Essen-Möller's criteria for delimiting his corresponding "nuclear" or typical group were mainly negative—exclusion of any with suspicious signs of schizophrenia—but even if this led to the inclusion of involutional psychoses which Kallmann would have excluded, it would have tended not to increase but to lower the age-standardized marriage and fertility rate. Similar considerations apply to Stanstedt's material. Whether the relative smallness of the sample of manic-depressive propositi in Kallmann's series could account to any degree for the discrepancy I am unable to judge, but it seems unlikely. In this dilemma, the prudent course seems to be to give more weight to the series which is larger and which can be followed up over a longer period. I believe, however, that Dr. Ødegaard will tell us of some relevant, fresh material, more in keeping with Dr. Kallmann's finding.

Since the bulk of the data, apart from Dr. Kallmann's, derive from mental hospital patients, I thought it useful to take the total manic-depressive clientele of the Maudsley Hospital, where the number of outpatients diagnosed as manic-depressive is one and a third times the number of inpatients with this diagnosis: in other words, many mild forms of the disease, which would not reach a mental hospital, are picked up in our outpatient department. Taking inpatients and outpatients together, there were, during the period 1949-1954, (Blacker & Gore, 1955; Blacker, 1957) 172 manic-depressive patients who had married between 1920 and 1929, and whose marriage had not been broken, by death or otherwise, before the woman reached the age of 45. It is evident from Tables 1 and 2, and Figure 1, (for which I am much indebted to Miss N. Goodman) that these patients, predominantly belonging to the Registrar General's social classes 2 and 3, did not differ in their fertility from the general population as recorded in the Family Census of the Royal Commission on Population. Nor is this a misleading comparison if the whole population of manic-depressives, single as well as married, are regarded. In the total sample of manic-depressive inpatients and outpatients at the Maudsley Hospital the proportion who were single, in the age-group 45-54 years, was practically the same, both for men (13 per cent) and for women (24 per cent), as for the population of the County of London, in which most of the patients lived (though appreciably higher than for the whole of England and Wales, where 9 per cent of men and 15 per cent of women of this age group are single).

There is, then, still room for doubt as to whether we have any striking quantitative departures to account for. Dr. Ødegaard has, however, drawn attention to some points, which I hope he will elaborate in the ensuing discussion. He has, for example, in his illuminating papers on this topic, (Ødegaard, 1946,

MANIC-DEPRESSIVE PATIENTS (INPATIENTS & OUTPATIENTS) DISCHARGED DURING 1949-1954

Table 1 MARRIAGES OF 1920-1924

No. of children born alive	*No. of patients* Men	Women	Total	%	*Cumulative* %	*Expected no. from all family census figures*
0	4	4	8	11	11	10
1	9	11	20	28	39	17
2	11	12	23	32	71	22
3	4	8	12	17	88	11
4	3	–	3	4	92	6
5	4	1	5	7	99	4
6	–	–	–	–	99	}
7	1	–	1	1	100	} 3
8	–	–	–			}
	—	—	—	100		
	36	36	72			

Table 2 MARRIAGES OF 1925-1929

No. of children born alive	*No. of patients* Men	Women	Total	*Cumulative* %	*Expected no. from all family census figures*
0	8	14	22	22	17
1	12	21	33	55	26
2	8	13	21	76	26
3	6	7	13	89	15
4	2	3	5	94	7
5	1	1	2	96	4
6	–	1	1	97	}
7	–	1	1	98	} 5
8	–	2	2	100	}
9	—	—	—		
	37	63	100		

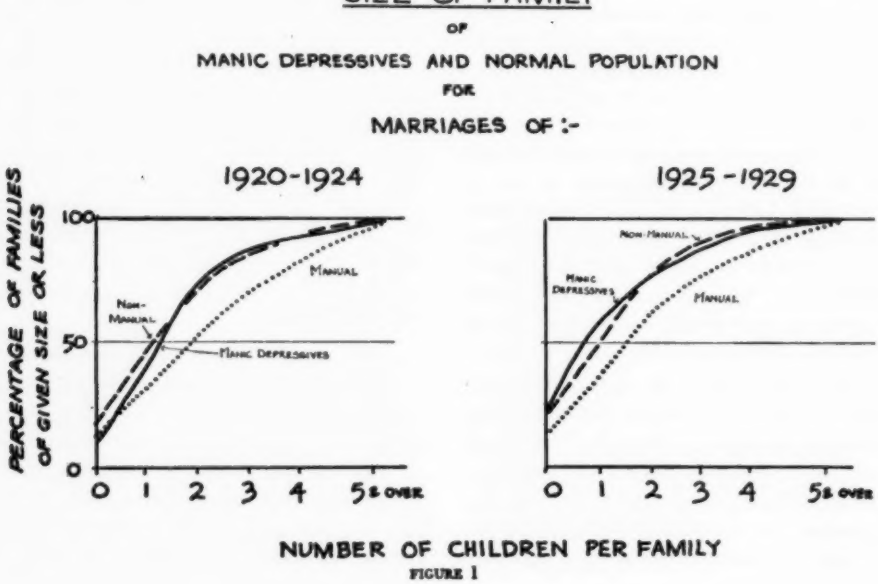

FIGURE 1

1953) speculated about why the proportion of single persons is higher in men than in women in every diagnostic group except the senile psychoses; but his data show that though this is true for the total manic-depressive sample, it does not hold good for all the separate age-groups; the reverse relation, in fact, is found in the manic-depressive groups aged 31-40 and 51-60. Still closer to our present theme is his finding that in the prepsychotic personality of male patients sensitive and depressive types are twice as common among the married as among the single: "evidently their serious outlook on life, coupled with considerable depth of feeling and craving for human contact, are factors which are predisposing to marriage." In women, on the other hand, the trend seemed the other way. He also found that nearly half (48 per cent) of the manic-depressive men who were single had at some time been engaged to be married; so had over a third (37 per cent) of the manic-depressive women. As my colleague, D. L. Davies (1956), has shown, engagement tribulations often bespeak severe disorder in the emotional development of the personality.

There is further the question of whether marriages contracted by manic-depressives endure. The figures from our hospital (Blacker 1957) show that for the triennium 1952-1954, 82 per cent of manic-depressive patients who had ever married were still married when they first attended the hospital, and that, of these more or less stable marriages, the average duration was 23 years for the manic-depressive men (of whom there were 377) and 21 years for the women (of whom there were 536): the average number of children born to these patients was 1.9. The divorced and separated, who represent some of the failures of marriage, amounted to only 6.4 per cent of our male ever-married manic-depressive patients, and 6.3 per cent of the women: whereas for schizophrenia the corresponding figures at the Maudsley were nearly twice as high (11.5 per cent and 12.6 per cent), and for hysteria and abnormal personality appreciably higher, as you would expect. Similarly in Dayton's Massachusetts patients (Dayton 1940), only 5 per cent of the ever-married manic-depressives were divorced or separated. However, Ødegaard, treating similar data from a rather different standpoint, found that the standardized admission rate for divorced manic-depressive men in Norway between the ages of 30 and 69 was 101 (as against 86 for the still-married patients) and for divorced manic-depressive women of that age group 166 against 117 for the still married). This he attributes not to the effect of divorce upon the patient's mental health but rather to the disturbing effect of the patient's personality and illness upon the maintenance of a harmonious marriage. (Ødegaard 1953)

Manic-depressive patients tend as a group to have physical and mental characteristics that are compatible with normal mental health but which are more often found among these patients than in the general population. Their "cycloid" temperament and their pyknic habit of body might well affect their choice of spouse, though in what way is a matter for conjecture: even if we subscribe to the "complementary needs" theory (Winch 1955), it is by no means clear what qualities in a mate would most gratify the needs of cyclothymic persons. However, a crude test of whether homogamy prevails can be applied by seeing whether those husbands and wives

(of manic-depressives) who themselves develop a psychotic illness show in that illness a manic-depressive picture. There are three collections of cases where both husband and wife became mentally ill—Schulz's, Elsaesser's, and my own, (Schulz 1940, Elsaesser 1952, Lewis 1957) totaling in all 174 couples—but in none of the three series was there a significant difference, in the cases of the manic-depressive propositi, between the number of their spouses whose illness was schizophrenic and the number whose illness was manic-depressive. Assortative mating doubtless occurs, but I do not know of any satisfactory evidence on this. Nor do we know what effect community attitudes towards the different forms of mental disorder have in determining choice of partner, once illness has become manifest in an otherwise eligible man or woman. In some families the attitude is such that their members would not be willing to marry anyone who had had treatment in a mental hospital, whereas in other families the prejudice against a patient showing the aftermath of schizophrenia would be strong but there would be no objection to the apparently fully recovered manic-depressive. The "mating" habits of manic-depressives could be altered by changes in hospital policy regarding community care for psychotic patients and by changes in public attitudes because of health education, propaganda and myths: but here again we cannot say whether there has in fact been a change on this account.

Essen-Möller has demonstrated that, so far as fertility is concerned, changes in the reproductive behavior of the general population are closely reflected in the reproductive behavior of the manic-depressives. It may fairly be assumed that these patients will also be responsive to changing social forces, which influence the probability of marriage. They are not likely to alarm us, however, so long as the marriage and fertility rates of manic-depressives approximate to those of the population in which the patients live. This situation may change rapidly. Introduction of an effective oral contraceptive; more precise knowledge of the genetic probabilities in this psychosis; general acceptance of the much promulgated view that every mental illness is "just an illness like any other"; a therapeutic advance that would nip most attacks of mania or depression in the bud before they disrupt the tenor of the patient's daily life—any of these probable events might make a lot of difference in the fertility of manic-depressives.

Though it is not strictly germane to our theme today, the apparent effect of marital state upon the course of manic-depressive illness deserves a brief reference. There are significant differences between the single and the married manic-depressives of both sexes in respect of duration of hospital stay. This was strikingly evident in Dr. Norris's large series (Norris 1959). Not only are single persons more likely to enter a mental hospital with this illness, but once there they are likely to remain longer. In other words, the single stay ill in hospital longer than the married. This, of course, touches again on the King Charles' Head of so many genetic and epidemiological studies of mental illness: how far can conclusions drawn from mental hospital patients with a specified disorder be taken to hold good for all persons with that disorder. No one will deny that many persons with manic-depressive illness have it in such a form and in such circumstances that they never enter a mental hospital, and may never even

consult a doctor for it. Many psychiatrists would further maintain that there is not a definite boundary, but rather a continuous gradation, between manic-depressive psychosis and psychogenic depression or excitement. In the face of these diagnostic quandaries, only a dogmatist would be ready to lay down the law at present about the mating and fertility patterns in families with manic-depressive psychosis.

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*DISCUSSION**

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IN COMPARING psychotic patients and the general population with respect to reproductive patterns, one has to consider the following factors:

1. The time trend: During recent decades the birth rate has decreased in most Western European countries. In Norway, the average number of children per mar-

*Since this paper was presented, the author has studied an additional 3,002 married women admitted in 1936-45, with a total of 9,067 children. The fertility in marriage was found to be 78.3 per cent of normal, which indicates that the difference between the insane and the general population shows a decreasing trend. The relation of schizophrenia to manic-depression was the same as in 1946-55, the figures for fertility in marriage being 90.2 and 80.2, respectively.

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riage of more than 20 years duration was 5.79 in 1920, and 3.11 in 1950.

2. Geographical variations, with a higher birth rate found in rural districts: In Norway, the average number of children per marriage in 1950 was about three in rural areas and two in cities.

3. Socio-economic class differentials, with a higher birth rate in the lower classes: This difference has become smaller, and what remains of it is largely due to the rural-urban differential.

4. Age of wife at marriage.

5. Duration of marriage.

Since all these factors are interconnected, it is difficult to determine the influence of the psychosis or of prepsychotic states upon fertility.

The national register of patients in mental hospitals in Norway furnishes information about the number of children born to each patient at the time of first admission. While these figures are not quite complete, particularly in regard to children who died in infancy, their general trend from the time this statistical system was introduced in 1936 has been fairly consistent, so there is no reason to consider them less accurate than the census data for the general population. Neither is there any evidence for major inconsistencies connected with clinical diagnosis, if the various diagnostic groups are compared. Diagnosis may play some role in the older groups because older patients may tend to omit children who died in infancy.

The census data of 1920, 1930, 1946, and 1950 give detailed information as to the number of children per married woman, with age at and duration of marriage. Single, widowed or divorced women and married men are not included.

According to the 1950 census, the number of children in existing marriages increased from an average of 0.56 in the youngest age group to 4.08 in women over the age of 60. Since patients are now classified according to age on admission and clinical diagnosis, an expected number of children for each subgroup can be estimated from census data. The given figures indicate how many children these patients would have had if their average number of children per marriage had been the same as that of the general population.

During the ten-year period 1946-1955, there were 4,229 married women among first admissions to Norwegian mental hospitals. Their actual number of children was 8,289, and the estimated number 9,598—a reduction of 13.6 per cent for the time prior to first admission (see Table 1). According to Essen-Möller and Kallmann, the subsequent fertility of these patients is substantially lowered, but we have no information about the number of children of discharged patients.

While the age at marriage of our patients is not known, it might be higher than in the average population, which would account for the lower fertility. However, since there is no evidence to substantiate this supposition, the figures in Table 1 may be regarded as sufficiently reliable for comparing the diagnostic categories.

It may be noted that the relatively small group of psychotic women with mental deficiency proves an exception to the general trend in that the actual number of their children exceeds the estimated figure, apparently due to their lower socio-economic and intellectual levels.

At the other extreme are the alcoholic psychoses and general paretics, with less than half the expected number of children. Apart from being predominantly from urban areas, these women are evidently not the maternal type. The number of children is also low in women with epileptic psychoses and psychoses associated with other organic brain disorders. Many of these patients had physical ailments before becoming psychotic.

The high fertility rate in women with symptomatic psychoses may be due to the large proportion of puerperal cases in this group. Curiously, the fertility in senile and arteriosclerotic psychoses is definitely below average, probably because so many of these patients are from cities such as Oslo that have a relatively low birth rate.

As to the major question in this discussion,

we find that while the fertility rate in schizophrenics approximates the expected figure, it is considerably lower in manic-depressives (94.1 and 82.7 per cent, respectively). The difference is statistically significant, but it cannot be assumed that this variation is determined by biological factors intrinsic to the disease. For instance, schizophrenics fall into lower socio-economic strata than manic-depressives. However, our Norwegian statistics indicate that these differences are less marked in married than in single men and still less in married women, if classified according to the husband's occupation.

Occupational groups in which schizophrenia is more frequently represented than manic-depressive psychosis are those in which general census figures show a relatively high fertility. Wives of laborers, for example, have an average of 3.2 children per marriage (more than 18 years duration, 1950 census) and in this group schizophrenia is 1.8 times more frequent than manic-depressive psychosis. The corresponding figures for wives of clerks and salesmen are 2.5 children and a ratio of schizophrenic to manic-depressive psychosis of 0.92. This socio-economic stratification may account for some, but probably not all, of the difference between the fertility rates of schizophrenic and manic-depressive women.

With the geographical distribution of these two psychoses fairly even, the higher birth rate in rural districts is not likely to play a role. There may be a certain predominance of schizophrenia in Oslo and other major cities which would tend to lower the fertility figures.

Our figures for the number of children estimated from census data are of course corrected for age, since the mean age of married schizophrenic women on first admission is 41 years and 49 for married manic-depressives. However, it must be realized that the actual fertility period of both groups came before hospital admission—earlier for the schizophrenics than the manic-depressive women. An analysis of age-specific rates would help to clarify this effect.

The rate of reproduction depends on both the fertility in marriage and the marriage rate. The known marriage rate of first admissions to Norwegian mental hospitals, according to diagnostic groups, is expressed in Table 1 as per cent of the marriage rate of women of corresponding age in the general population. Multiplication of these percentages with the relative figures for fertility in marriage gives a rough estimate of the rate of reproduction.

Table 1 shows that the psychotic women as a group have a rate of reproduction of only 39 per cent as much as the general population. In this analysis the difference between schizophrenia and manic-depressive psychosis comes out very clearly. It is apparent that the total reproduction rate in the manic-depressive group is higher than in the schizophrenics, due to their much higher marriage rate.

The foregoing data do not include patients who marry and have children after their first admission to the hospital. It was shown in a previous report that 28 per cent of the married male patients and 18 per cent of the married female patients had married some time after the first onset of psychotic symptoms, mostly during a remission of the disease. The majority of these marriages, however, were contracted before first admission to hospital.

According to Essen-Möller, the relatively few who marry after having been in a psychiatric hospital are likely to have few children. These "remission marriages" hardly represent a major error, but they would undoubtedly bring the reproduction rates up somewhat by raising the marriage rates, particularly for manic-depressives.

Illegitimate children are not included in our figures, but they would make little difference inasmuch as they have about the same prevalence as in the general population. Altogether, they comprise 2.51 per cent of the total offspring as against 4.12 per cent in the general population, but our figure for the patients is probably too low, owing to incomplete information. Also, patients are inclined to regard as legitimate a child born previous to the marriage, while

TABLE 1
FERTILITY OF MARRIED WOMEN FIRST ADMITTED TO MENTAL HOSPITALS, 1946-1955

	No. of married women admitted (1)	*No. of children* Found in present survey (2)	Estimated from census data (3)	*Fertility rate* (4) [1]	*Marriage rate* (5) [2]	*Reproduction rate* (6) [3]
Psychosis with mental deficiency	53	127	103	123.1	19.6	24.1
Schizophrenia	658	1,249	1,327	94.1	26.8	25.2
Manic-depressive psychosis	473	1,009	1,220	82.7	75.5	62.5
Other functional psychoses (without gross pathology)	1,872	3,266	3,903	83.7	53.4	44.7
Symptomatic psychosis	207	387	396	97.7	105.1	102.7
Epileptic psychosis	27	45	58	78.0	36.4	28.4
Alcoholic psychosis	26	40	66	60.6	250.0	151.6
General paresis	51	60	138	43.6	129.5	56.4
Senile and arteriosclerotic psychoses	326	1,003	1,139	88.1	49.8	43.8
Other organic psychoses	74	166	213	78.0	67.7	52.8
Unclassified cases	106	213	218	97.4	126.1	122.6
Without psychosis	356	724	817	88.5	57.9	51.2
All diagnoses	4,229	8,289	9,598	86.4	45.2	39.1

[1]Per cent of observed over estimated (column 2 over column 3, times 100).
[2]From census data and overall hospital records.
[3]Fertility and marriage rates multiplied.

the census data are based strictly upon the marital status of the woman at the time the baby is born. Our figures indicate that among the single patients one in seven were reported to have had a child previous to admission.

The magnitude of the social and eugenic problem associated with these children of psychotic mothers is illustrated by the figures in Table 2. The 9,378 first admissions dur-

TABLE 2
NO. OF CHILDREN BORN TO WOMEN FIRST ADMITTED TO NORWEGIAN MENTAL HOSPITALS, 1946-55
(ALL DIAGNOSES)

	Single	*Married*	*Widowed and divorced*	*Total*
No. of women	4,043	4,229	1,106	9,378
No. of children	274	8,289	2,347	10,910

ing the ten year period 1946-1955 had a total of 10,910 children, which means that nearly 1,100 individuals are added every year to the group of people who have a psychotic mother. Those with a psychotic father appear to be about equally numerous.

Fortunately, from the psychiatric standpoint the situation is somewhat mitigated by the fact that in more than a fifth of these cases the mother's overt psychosis occurred after the child was full grown and out of the home. From the genetic point of view, one has to take into consideration that not all of the conditions included in this table are genetically determined. Nevertheless, for the most part the eugenic problem remains a serious one.

Additional information from family investigations is seen in Table 3, which gives some data from the families of a representative group of 306 probands with a major functional psychosis. In the proband generation, the mean number of children per marriage was 6.93 and in the parents' generation 5.86. The reason the number of aunts and uncles was found to be lower than that of the siblings, despite the time trend, is probably that some of the children who died in infancy are omitted in the older group, while such instances are less likely to be forgotten among the siblings.

Since no satisfactory material of earlier data is available, the comparison with the general population of Norway has to be based on data from the 1920 census. The family investigation material goes somewhat

TABLE 3
FERTILITY IN PSYCHOTIC PROBAND FAMILIES

	No. of probands	*No. of sibs*	*Mean sibship size•*	*No. of parents and their sibs*	*Mean sibship size in parental generation*
Schizophrenic and allied psychoses	191	1,154	7.04	2,157	5.64
Manic-depressive and allied psychoses	86	482	6.49	1,025	5.96
Other psychoses without gross pathology	29	158	6.44	406	6.99
Total	306	1,794	6.93	3,588	5.86

•Mean number of children in "completed" Norwegian marriages (wife over 45 years of age, childless marriages excluded, 1920 census): 5.86.

further back, however, and consequently we have used for purposes of comparison a general population with too low a birth rate, perhaps.

Excluding childless marriages, the average number of children in so-called completed marriages (wife over the age of 45) was 5.86. However, not all marriages in the family material were "completed." A number of women died or lost their husbands before the age of 45. The number of children in the proband families should therefore have been somewhat higher. This error will to some extent cancel out the one mentioned above, and the conclusion seems to be that the fertility of the parents and grandparents of these probands does not differ substantially from that of the general population.

In this material the diagnosis was made by the author, in every instance, mostly from the case history. In the proband generation, the fertility seems to be definitely lower in the manic-depressive than in the schizophrenic group. The difference is moderate but gains in importance because of the similar trend which was found in the larger statistical material. Also, it agrees with Kallmann's findings in his twin material, while departing, as noted by Lewis, from the findings of other authors.

It is not surprising to find in manic-depressives a somewhat lowered marital fertility. A similar observation is made in the large group of "other psychoses without gross pathology" (paranoid states, reactive depressions, etc.). What is unexpected is the high fertility of married schizophrenics. The explanation is probably to be found in the prepsychotic history of these patients. In my opinion, preschizophrenic personality changes constitute a considerable marriage handicap. However, it does not follow that this factor also operates against having children once the marriage barrier is passed. Clinical material abounds in cases of uninhibited propagation due to preschizophrenic personality traits, and even a moderate number of such instances will suffice to increase the average number of children.

Fertility *per se* is not likely to be affected by schizophrenia or manic-depressive psychosis. The most significant factor in reducing reproductivity in both psychoses would seem to be the decreased marriage rate, with decreased marital fertility playing only a secondary role.

See Dr. Aubrey Lewis' bibliography on page 137 for references.

MATING AND FERTILITY PATTERNS IN FAMILIES WITH SCHIZOPHRENIA

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SCHIZOPHRENICS PROVE TO HAVE A very different number of children in samples from different countries, from urban and rural areas, from different social groups, and at different times. This variation is apparent from a comparison and a subdivision of samples investigated by Kallmann (15), Böök (3), and myself (9), coming from Berlin, North Sweden, and Bavaria, respectively. It is therefore mandatory, first, to study the reproduction of schizophrenics in relation to the population from which they originate.

There is a second requirement. Suppose there are some findings characteristic of schizophrenia in relation to the respective populations. Obviously these might have been brought about by differential fertility in its proper sense, i.e., by a different birth rate specific to age-groups and to duration of marriage. But the same effect could perhaps have been produced by some peculiarity of age-distribution, which in turn could have been due to differences in mortality or rate of migration, or simply to the distribution of age at onset of the disease. Or, finally, the distribution of marital state might have played a role. Larsson (17) pays much attention to detecting such fallacies in the study of the interaction of population changes and heredity.

It is necessary, then, for any closer study to investigate each component of reproduction separately and their interplay, the mechanism of which has been very clearly demonstrated by demographers like Dublin and Lotka (7) of the Metropolitan Life Insurance Company of New York and the late Professor Wicksell (33) of the University of Lund.

Considering legitimate fertility first, a close study revealed that samples of schizophrenics exhibited the same trend in differential fertility and decline in births as generally seen in Western communities. However, it appeared that schizophrenics followed suit somewhat more slowly: thus male schizophrenics seemed to start from about the same level as the population with a high birth rate, and subsequently to lag behind in the general decline in births. Female schizophrenics apparently started from a lower level, and the further reduction in their fertility was more or less neutralized by the more rapidly declining population level. The average reproduction of both sexes shifted, as the general birth rate gradually declined, from a level lower than the population to a level slightly higher.

It is difficult to state whether this finding of a delayed decline of birth rates in schizophrenics has general validity or is typical only of the Bavarian sample investigated (9). In any case, there would be reason to assume that such phenomena are transitory. Thus in a population where birth control had been practiced for a long time, it seems reasonable to assume that the legitimate fertility of schizophrenics should be about equal to that of the population.

The same would probably be the case with illegitimate fertility.

These observations all pertain to the time prior to the first admission to a hospital. Post-psychotic fertility may be estimated, in marriages contracted before admission, at about 70 per cent of the pre-psychotic fertility, and this decrease is apparently not ascribable to hospitalization only. In marriages contracted later, there was no such demonstrable reduction.

It is possible, then, that pre- and post-psychotic fertility taken together, as judged by age-groups and duration of marriage is somewhat reduced in schizophrenics. This was also Kallmann's finding (15).

In regard to marital state, it is well known that there is a much higher proportion of unmarried people among schizophrenics than otherwise in the population. This phenomenon attracted attention long before the concept of "schizophrenia" or even of "dementia praecox" had come into use. It has been studied in many countries, not the least recently in the United States (2), (4), (5), (6), (9), (13), (15), (21), (22), (25), (29), (30), (31).

This over-representation of single persons among schizophrenics is present in all age-groups of schizophrenics as early as the time of the first admission. In fact, the marriage rate of the index cases-to-be was shown to have been constant at its low level throughout their pre-psychotic life and is therefore not a product of the manifest disease, as formerly believed. It could rather be argued that it is due to some traits of personality. A third hypothesis is that married life in itself could protect persons from schizophrenia. There is also a fourth possibility, namely, that unmarried schizophrenics are taken care of by their families to a lesser extent than the married and are thus more frequently hospitalized.

Let us consider this hypothesis of excess hospitalization. In the absence of an exact measurement I once tried instead to investigate widowers and widows, since these can be expected to have the same personality as the married (9). Widowers and widows proved to have been admitted on an average twice as often as the married, and about the same proportion was present in a sample presented by Dayton (5). Accordingly, a correction was attempted in doubling the number of married, and computing the frequency of unmarried in the sample thus enlargened. The surplus of unmarried schizophrenics over the normal level is thereby noticeably reduced but still remains considerable. While the uncorrected distribution of unmarried corresponds to a specific marrying rate almost three times smaller than normal, the corrected distribution corresponds to roughly half the normal marrying rate. (Assuming a stable population, half marrying rate results in a distribution of unmarried persons where every age-specific frequency is the square-root of the normal frequency, and a marrying rate three times reduced results in cube-root frequencies (33)).

Later I had at my disposal a sample investigated at the Rüdin Institute consisting of brothers and sisters of schizophrenics who had been followed up in regard to marital state (10). Their distribution fell, as expected, between the schizophrenics and the general population. However, the sisters were so close to the corrected distribution that one might suspect the correction to have been overdone. Now the siblings included altogether 146 schizophrenics who were

identified through family investigation independently of admissions. Among these, the single were admitted, on an average, only 1.2 times (instead of twice) as frequently as the married. Possibly, then, the correction factor for hospitalization should not be greater than this.

Accordingly, the over-representation of unmarried persons at first admission is to be regarded largely as a true finding. Professor Ødegaard (25), who studied very closely the over-representation of single persons, argued against the hospitalization theory. He also dismissed the hypothesis of protection by marriage as a main factor and stressed the importance of personality. Of special interest is his inquiry into the personality of married and unmarried mental patients. The traits likely to be responsible for not marrying are, as pointed out by Norris (24), about the same as those which are considered signs of a poor prognosis, and Norris showed that single schizophrenics stayed longer in hospitals than married ones.

In samples from different countries the over-all frequency of single persons among schizophrenics stays rather constant at 70 to 80 per cent in males. In females there is a variation from this level down to 40 to 50 per cent in some American samples (21), (31), and even as low as 27 per cent in a community of Northern Sweden investigated by Böök (3). Similar though far more subtle differences were noted by Ødegaard (25) between rural and urban areas in Norway. These authors were inclined to interpret their findings by referring to social factors and to the shortage of women in the respective districts. Female schizophrenics would thus be more susceptible to social influences than males, perhaps because of the more passive part played by them in reproduction.

After admission to hospitals, the marriage rate is still more reduced, and, as is the case with fertility, this is by no means a consequence of hospitalization exclusively. The average marriage rate after admission in my German sample amounts to something like one-sixth of the normal.

Consideration should also be given to the problem of mate selection in schizophrenics. To my knowledge there are three systematic investigations into the families of mates, two of which report a higher incidence of schizophrenia (8), (12), (18). Standard errors, however, are comparatively great, so that a deviation from normal expectancy can hardly be considered to be established. This would indicate that mate selection in schizophrenics may be reasonably normal.

To secure an expression of the total reproduction of schizophrenics in relation to the population, one should now recombine, in a standardized manner, the distributions of fertility and of marital state which so far have been studied apart. Also a certain excess mortality has to be taken into account, but its importance is not very great any more than is the reduction in fertility proper. The predominant factor here is the heavy over-representation of unmarried people. Age at onset, of course, plays a part but not so much as one might be inclined to think, because the marriage rate stays at a low level even prepsychotically and appears to do so from the very beginning of adult life.

All factors considered, using the figures from the Bavarian sample and putting the correction factor for hospitalization at 1.2, the ensuing total

number of children of schizophrenics may be estimated at about 40 per cent of that of the corresponding population. From these 40 per cent, again 30 to 40 per cent refer to children born after the onset of the disease.

This result would seem reasonable since, by applying the following procedures, we can actually check it. Putting the incidence of schizophrenia in the population at 1 per cent and in children of schizophrenics at 10 to 15 per cent, the above-mentioned reproductive rate of schizophrenic index cases amounting to 40 per cent would mean that some 8 to 12 out of a hundred schizophrenics descend from a schizophrenic parent. As for the parents themselves, 4 to 6 per cent would be schizophrenic, which is in good agreement with the majority of the findings. In Böök's sample (3), where the incidence in parents is exceptionally high, there is also a higher reproduction in the index cases.

A reversal of this checking procedure was indicated by Koller (16). According to Mendelian rules, the incidence in parents of index cases with any hereditary disease, irrespective of its mode of inheritance, should be equal to the incidence in children. If, then, the incidence in children is 10 to 15 per cent and in parents only 4 to 6 per cent, the discrepancy is accounted for by assuming a reproduction reduced to 40 per cent of the normal, which is the reduction actually found in our schizophrenics.

A third possibility of checking is the following (11). Parents do not really enter into statistical observation until they become parents of the later index cases. Before this time they were subject to selection in the sense that their contemporaries who fell ill with schizophrenia were excluded to a certain extent. If nobody could have a child after becoming schizophrenic, then the cases of schizophrenia which can still be found among the parents of schizophrenics must have occurred later than the birth of the index case. Therefore, obviously it would be misleading to relate schizophrenia in parents to the total period of risk covered by them from 15 years onward. Instead, the observation period should be counted from the age at birth of the later index case. In fact, in a Swedish sample of parents of schizophrenics, their observation time, when reduced according to this principle, was only one-fourth of the period usually used for calculation. A modification has to be made to allow for the fact that schizophrenics are not completely excluded from reproduction after falling ill, and to account also for their reduced reproduction before onset of the disease. With this modified correction, the incidence in parents actually becomes about the same as in children, which may be taken as an indication that the principle of correction is right. The magnitude of the correction itself corresponds to the estimated reduction in reproduction and may thus serve to check this estimate.

Throughout this study, the group of schizophrenics was treated as an entity and as though diagnosis was everywhere uniform. Some attempts at differentiation should now be mentioned. Hebephrenics and catatonics,—males at least,—were stated to have a lower frequency of marriage and apparently also a smaller number of children than paranoids (9), (15). Schizophrenics with initial symptoms like those of manic-depressive psychosis were found to have married as frequently as manic-depressives before

falling ill, but as rarely as schizophrenics in the later course of their illness (9). There was a suggested indication that schizophrenics with a "rheumatic" history, who were also preferably non-paranoid, would comparatively more often be prevented from becoming parents, especially fathers, owing to early onset of the disease (32). A subdivision in recovered and non-recovered gave contradictory results for the distribution of marital state in the two sexes (9). In marriages contracted after the onset of illness,—thus presumably in recovered patients,—there was no demonstrable reduction in fertility (9). Schizophrenics from consanguineous matings were stated to have married more rarely than other schizophrenics and to have had fewer children if married (23).

Applications of the findings should now be considered. It may be of interest to remember that the reproduction rate of schizophrenics has played a role even in the theory of psycho-dynamic causation of schizophrenics. Alanen, in his recent book (1), stressed a fact already mentioned by Penrose in quite a different context (27), namely, that mothers of schizophrenics are more often schizophrenic than are fathers—usually about twice as often—and in Alanen's sample still more frequently. Alanen felt that this discrepancy could probably only in part, be accounted for by sex differences of fertility, and so interpreted it instead in terms of a disturbed mother-child relationship. In my opinion, sex differences of reproduction would probably account for the entire discrepancy. In Böök's sample (3), reproduction in males is about three times lower than in females. Even if this proportion is likely to have been less pronounced in the preceding generation, like the shortage of women which is supposed to have been instrumental in producing it, it would probably be enough to make up for the discrepancy actually found. In my own Swedish sample of parents of schizophrenics (11), the above-mentioned reduction of the period of observation, which was seen to be an expression of the reduction in reproduction, was also three times greater in fathers than in mothers. So much for the psycho-dynamic aspect.

Turning finally to the genetic applications, I may refer to Professor Aubrey Lewis, who in last year's Galton Lecture made an excellent survey of fertility and mental illness (19). He traced the problem as far back as a century ago and very clearly stated the changing views from the practical eugenical standpoint. Professor Lewis also demonstrated the shift toward study of theoretical problems, such as why schizophrenia remains as prevalent as ever. To answer this question, inquiries were made a long time ago into the marriage rate and fertility of relatives (9), (15), (20). No evidence emerged to support the idea that above-normal reproduction in relatives would compensate for subnormal rates in the schizophrenics themselves; the contrary was rather the case.

It was therefore only natural that, in the search for an explanation of the unchanged prevalence of schizophrenia, mutations should be investigated: estimates of their frequency were attempted, particularly by Penrose (28) and Böök (3). Another line of theoretical research was a study of differences in maternal age at birth of male and female schizophrenics. This study was conducted by Goodman (14) upon a suggestion from Penrose (26) who touched upon subtle questions connected with the theory of

the evolution of dominance by natural selection.

To conclude, the exposition here given will have shown that with regard to both main aspects of our problem, "facts" as well as "applications," the trend was from a comparatively simple and practical approach toward a more complex and theoretical one. It can therefore be maintained that the reproduction of schizophrenics is today as intriguing a problem as ever.

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ERRATA: *In the article "Breeding Structure of Human Populations" by M. Fraccaro (Eugenics Quarterly, 6, No. 1, March 1959) Figs. 2 and 3 should be interchanged.*

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